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(Complete contents inside)

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ARCHIVES OF OPHTHALMOLOGY.

EXFOLIATION OF THE LENS CAPSULE.

BY DR. ARTHUR N. ALLING, NEW HAVEN, CONN.

(With one illustration on Text-Plate I.)

BERGER in 1882 demonstrated that the lens capsule could be separated into lamellæ when treated with permanganate of potash and since that time others have confirmed his observations. During the past three years I have seen with the slit lamp seven cases of a peculiar change on the anterior capsule of the lens which evidently has some direct bearing on this subject. The appearance to which I refer consists of a slightly opaque disc on the center of the anterior surface of the lens with a white ragged looking border. The disc is a little smaller than the average size of the pupil. Peripherally from its edge is a clear black zone where the capsule seems to be transparent. Outside of this and only to be seen with a dilated pupil are patches composed of white dots resembling frost. On the pupillary edge of the iris in most of the cases there is a row of white tags rather regularly spaced and apparently loosely attached to the pigment border. The obvious explanation of these cases is that a superficial lamella of the anterior capsule has degenerated and become friable so that the normal movements of the iris in the contraction and dilatation of the pupil have denuded the rest of the capsule which remains clear, leaving the slightly opaque disc in the very center. Particles of the membrane thus detached have become fastened to the pupillary edge and some shreds

have been freed in the anterior chamber since particles have been seen on the posterior surface of the cornea. Under the iris the membrane has become partly detached and partly remains in the form of granular patches. The fact that only certain areas of the capsule under the iris have been detached is probably due to the uneven character of its posterior surface. A somewhat different appearance was found in one case where the lamella had been detached in sheets (Case 5). If the assumption is correct that particles of membrane have been loosened might it not be possible that they have floated with the current into the iris angle and serve to lessen the outflow, for glaucoma has accompanied some cases? In every one of my cases a cataract was present in varying stages of development. In one instance where the cataract was extracted a piece of the capsule was preserved, as also the iris excised in the iridectomy, but unfortunately the examination yielded no definite results as the iris was naturally much traumatized and the capsule was very difficult to study.

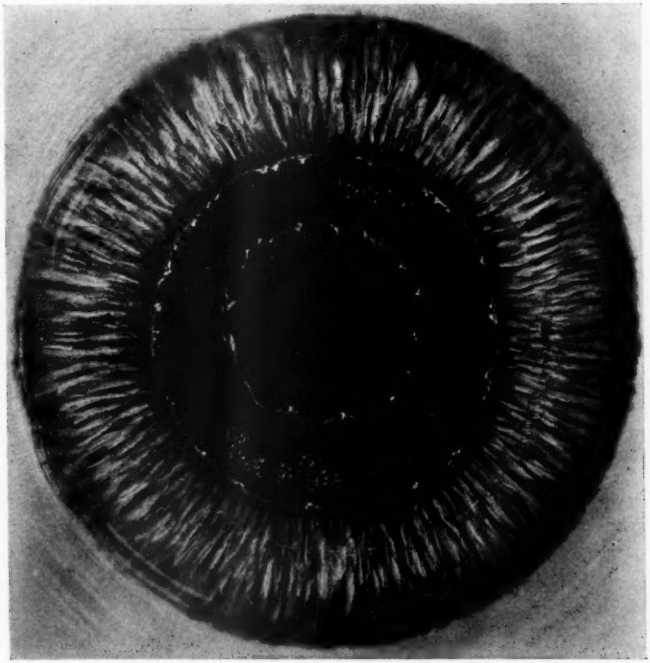
Examination of literature shows that Vogt has described twelve cases in general of a similar nature, nine of which had chronic glaucoma. Subsequent observations show that this is by no means the rule. At that time he does not seem to be satisfied as to the exact explanation of the condition. Rotter, Meesmann, Goulden, Wollenberg, and Bedell have reported similar cases, and Vogt, Elschnig, Kraupa, Kubik and others have noted exfoliation of the capsule in glass-blowers.

Following are some of the slit lamp notes on my own cases:

(1) Mrs. L. W. Caviner, aged 60. Immature cataract R., incipient L. In both eyes there is a faint disc occupying a central area and there are minute dots on its surface. The edges are quite rough and in some places are curled over and crumpled. Peripherally there is a perfectly clear zone. Outside of this again are patches of granular deposits of white dots. On the pigment of the pupillary edge are small delicate masses of white material like little tags of cotton arranged at more or less regular intervals. Pupillary edge is translucent by transmitted light from lack of pigment.

(2) Mrs. Bridget Moran, aged 80. R. incipient cataract, L. mature cataract. Both eyes have scant pig-

ILLUSTRATING DR. ARTHUR N. ALLING'S ARTICLE,
"EXFOLIATION OF THE LENS CAPSULE."



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ment at pupillary edge and tags all around. The anterior capsule shows shreds of white membrane with a circular arrangement and whole capsule is covered with small vacuoles. On the posterior surface of the cornea there are fine deposits resembling those at the iris edge. Extraction and unsatisfactory examination of the capsule and iris.

(3) Charles McCarthy, aged 75. Incipient cataracts in both eyes. In pupillary area there is a distinct slightly opaque disc like thin membrane limited by a ragged border which in some places projects and curls forward like little tongues. There are tags at pupillary edge.

(4) Mary Sullivan, aged 56. Incipient cataract R. and L. In right eye anterior surface of the lens has a few pigment spots and also some clearly defined shreds in nearly regular circle. Periphery with the dilated pupil shows the capsule to be covered in places with fine granular opacities.

(5) J. S. Monagan, aged 74. Immature cataracts R. and L. Right preliminary iridectomy. In right eye anterior capsule has grayish appearance and at the temporal edge of the coloboma there is a sheet of delicate membrane which folds over on itself like a scroll and has a few pigment spots on it. There is also a sheet of membrane below hanging into the anterior chamber over the edge of the iris. Left eye shows a scroll of very thin membrane attached to the lower edge of the iris and it waves with the iris movement. Looks as if it had been torn off the capsule on the nasal side.

(6) F. P. Noera, aged 73. Chronic non-inflammatory glaucoma, sclerosed cataract. In right eye there is iris atrophy at the edge. On the anterior capsule a gray disc with frayed edges, outside this a clear zone and beyond patches of very fine dots. The impression is that the anterior capsule has been torn off except in the center, numerous small patches being attached at the pupillary edge. In the left eye, pupil not dilated, but tags at iris border.

(7) Steve Daddero. This case is one of my associate's, Dr. Yudkin, and the findings are typical as described above.

The picture which is presented in these cases does not appear to be the result of an inflammatory process since there is absolutely no evidence of activity and there are no syn-

echiæ or pigment masses on the anterior capsule giving evidence of any previous disease, moreover the central disc is very evidently a thin homogeneous membrane not like an exudate. On the other hand it is probably not congenital because the little white masses are at the very edge of the pupil while a persistent pupillary membrane is attached to the shelf which divides the inner and outer zones of the iris and furthermore the condition has never been seen in young persons. In view of these considerations the following conclusions seem justified:

1. The membrane is not an exudate but an exfoliation of the anterior lamella.
2. The condition is a senile change although it has been observed in glass-blowers, who are exposed to infra-red rays.
3. Glaucoma is not present, as a rule, contrary to Vogt's observation but there is usually a cataract.
4. The formation of the central disc and its ragged border as well as the deposits at the pupillary edge are due to the denuding of the capsule outside the central disc by the iris movements.
5. Sometimes the exfoliated lamella comes off in sheets instead of flakes.

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ENDOPHTHALMITIS FROM RETAINED FOREIGN-BODY PHACOCYST.

By Dr. DERRICK T. VAIL, Jr., Cincinnati, Ohio, with a Pathological Report by Dr. F. H. VERHOEFF, Boston, Mass.

(*With three illustrations on Text-Plate II.*)

THE following case report illustrates that a harmless inflammatory cyst surrounding a foreign body, located in the substance of the crystalline lens, can, if allowed to remain within the eyeball after extraction of the foreign body, precipitate a violent and destructive inflammatory reaction. So far as I have been able to determine no similar case has been hitherto described.

C. H., 38, a mechanic and truck driver, entered March 30, 1926, complaining of loss of vision in the left eye for the past two weeks.

History.—Two weeks ago he noticed a "film" spreading over the sight of the *left eye.* He consulted a physician who told him he had a cataract. The vision gradually became poorer, accompanied by slight twinges of pain, until he could but distinguish light. Before this the left vision was good. No history of an accident or injury to the left eye could be obtained.

Four months before, while pounding a gear pin with a hammer some foreign particles flew up and struck him in the *right eye only.* Pieces of dirt were removed by the local physician and drops instilled. The eye cleared up and gave him no further trouble.

P. E. O. D. Normal in every respect, vision = $\frac{20}{20}$.

O. S. Slight ciliary congestion. The cornea was clear except for a small linear scar, *2mm* long, immediately below and temporally to the pupil. The pupil was semi-dilated and did not respond to light. (He had had atropin

instilled by local physician.) The lens was cataractous and immediately beneath the scar on the anterior surface of the lens were seen a few pigmented particles. Vision = $\frac{18}{200}$. He located the light in all parts of the field. Tension = 18 (Schiötz). Fundus could not be seen.

Slit lamp examination, left eye.

The cornea was normal except for the linear scar mentioned above. This was a narrow homogeneous white opacity, without pigmentation. A rupture of Descemet's membrane was looked for, but not seen. The lens was diffusely opaque and "asbestos" colored. A few round and oval vacuoles were noticed in the epithelium and anterior cortex. Immediately beneath the corneal scar in the anterior capsular epithelium were several small vacuoles, and the anterior cortex in the immediate neighborhood was especially opaque. The iris was apparently normal. A diagnosis of an intra-ocular foreign body and secondary cataract was made.

An X-ray picture was taken and the foreign body was revealed. It measured 1 x 1 x ½*mm*. Its position was 4*mm* below the horizontal plane, 3*mm* temporal side vertical plane, and 8*mm* back of center of the cornea.

The giant magnet was used to pull the foreign body into the anterior chamber, where it lay in the floor as a small glittering particle.

The patient was unprepared to stay in the hospital at that time, but returned two days later (April 1, 1926) for the extraction operation. At this time the eye was not congested. A small keratome incision was made in the limbus above and the foreign body extracted without difficulty, with the hand magnet. Atropin 4% was instilled and the eye bandaged. No iridectomy was performed.

For two days following the operation there was no more local reaction than one would expect. On the third day the congestion was greater. The wound was in good approximation and appeared healthy.

April 7, 1926. A small hypopyon (1*mm*) appeared. The globe was deeply congested and the patient complained of a severe pain. The cataract was unchanged in appearance, the light field was normal and the tension measured 12 (Souter). Feeling that a lens protein reaction was taking place, a lens protein test was done. This was negative.

The next day a small, round, yellowish vesicle appeared in the anterior chamber, apparently attached to the lens at the spot where the foreign body had been pulled through. It gave the appearance and color of a miniature grapefruit. The hypopyon now measured 3*mm*. The eye was

ILLUSTRATING THE ARTICLE OF DR. D. T. VAIL, JR., "ENDOPHTHALMITIS FROM RETAINED FOREIGN-BODY PHACOCYST."

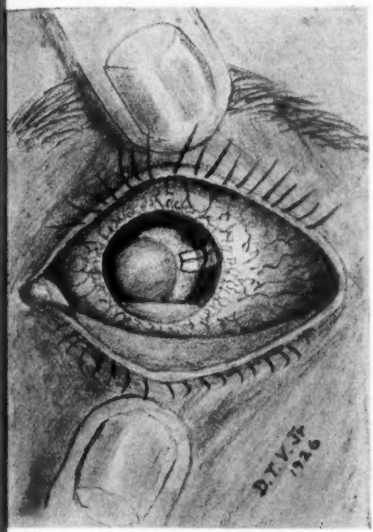

Fig. 1. Appearance of eye one day before operation.

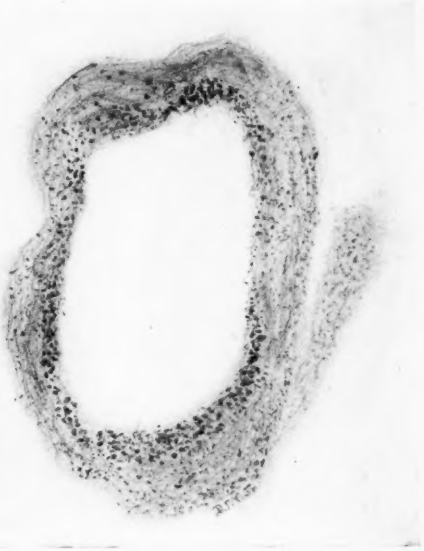

Fig. 2. Low power sketch of the cyst wall.

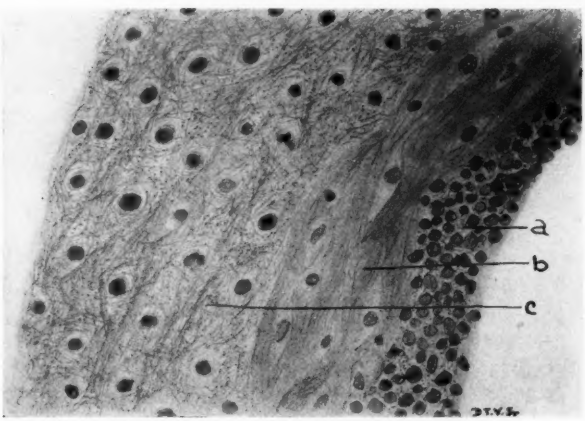

Fig. 3. High power sketch showing:
(a) Necrotic endothelial phagocytes.
(b) A few broken up lens fibers.
(c) Fibrin and pus cells.

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deeply congested, and the cornea cloudy. The wound had firmly united and there was no more congestion here than in other parts of the eye.

April 10, 1926. 10*cc* of sterilized milk injected intramuscularly. A good general reaction ensued, but the eye appeared worse.

April 12, 1926. Another 10*cc* of milk injected. But no effect on the eye. The small yellow vesicle had increased in size, and the cornea was more cloudy. The light field was still normal, and the tension = 12 (Souter).

April 13, 1926. Following an iridectomy the cataract was extracted, and the anterior chamber syringed with normal saline. Fortunately the sac wall of the yellowish vesicle was obtained, although the contents, purulent in nature, were lost. Some material from the anterior chamber was obtained under sterile precautions and used for culture and animal studies. *All the cultures were negative.* Material injected intraperitoneally in a guinea pig was without effect. Some material was also injected into a rabbit's lens with no result except for a traumatic cataract.

The eye cleared up miraculously. The bulbar congestion faded and the wound healed without any complication.

April 27, 1926. Patient discharged. The eye was white, the cornea clear, and the pupil black. There was a very small amount of cortical substance remaining in the pupillary area on the nasal side. Vision with a + 12.0 D. sph. ⊂ + 1.0 cyl. ax. 30 = $\frac{20}{20}$. Adding + 3.50 D. sph. patient read Ja. #1.

The vitreous showed a few string-like floating opacities, the fundus, field of vision and tension were normal.

August 10, 1926. The patient was examined and the following notes were made. Left eyeball not inflamed a particle. Cornea clear except for the scar. One can see the anterior body of the vitreous behind the iris by focal illumination. No floating opacities seen. Vision, + 12.0 D. sph. ⊂ + 1.0 cyl. ax. 15 = $\frac{20}{20}$.

The "cyst" wall, measuring 2 x 4*mm* in diameter, preserved in a 10% formalin solution, was sent to Dr. F. H. Verhoeff, Massachusetts Eye and Ear Infirmary, for pathological examination. His report is as follows:

"The specimen consists of a thick wall surrounding an empty space. The wall consists of soft lens matter infiltrated with endothelial phagocytes, necrotic cells which cannot be identified with certainty, and, externally, a thick layer of fibrin with a moderate number of pus cells. The tissue probably originally formed around the foreign body and after being displaced forward caused a moderate inflammatory reaction with deposition of fibrin."

Comments.

To me the most interesting feature of the case was the extraordinary clinical inflammatory reaction caused by the displaced sac. To all ordinary appearances the eye was doomed. The hypopyon, the tremendous congestion, the œdema of the eyelids, and the cloudy cornea pointed to a rapid destruction of the eyeball. Drastic measures were indicated. Since it was felt that, in spite of the negative lens protein test, there was an endophthalmitis phaco-anaphylactica going on, it was considered wise to remove the irritating substance. Results justified this judgment.

So far as I can determine no other case in which the cyst surrounding a foreign body caused an inflammatory reaction after extraction of the foreign body has been reported. It seems strange that containing the foreign body it should be tolerated without inflammation; but after removal of the foreign body, the sac and its purulent contents should in turn act as an irritating substance, is indeed unusual.

The slit lamp in this case was valuable in proving without doubt that the cataract was due to an intraocular foreign body. Siderosis of the lens could be definitely diagnosed by its use. The case emphasizes once more that a unilateral cataract in a young man, especially in the absence of a history of injury, must be carefully studied before ruling out the presence of a foreign body.

Summary.

A young mechanic presented himself with a unilateral cataract and no history of injury. Examination with the slit lamp revealed the track of a particle of iron, and the diagnosis of an intra-capsular foreign body was confirmed by X-ray. Magnet extraction of the foreign body by the anterior route was followed by the appearance of a yellow cyst attached to the anterior surface of the lens at the exit focus of the foreign body, hypopyon and a marked inflammatory reaction of the eyeball. Removal of the lens and cyst resulted in recovery and nearly perfect sight. Animal and

cultural experiments were negative for pathogenic organisms. A skin test with lens protein material was negative. Specimens studied microscopically proved to be the capsule formed as a foreign body reaction.

ESSENTIAL PROGRESSIVE ATROPHY OF THE IRIS; A SECOND COMMUNICATION.

By Dr. GEORGE E. de SCHWEINITZ, Philadelphia.

(With five illustrations in the text and one in color on Text-Plate III.)

THE object of the present communication is to detail the case record of a patient with progressive atrophy of the iris, who has been under observation for fourteen years, and whose earlier clinical history was reported to this Society in 1915; to analyze the reports of similar cases which have appeared in the literature; and to refer to some of the theories which have been advanced to explain the development of the iris lesions.

The essay, therefore, is concerned only with a group, not a large one, of iris atrophies for which no definite cause has thus far been discovered, either as the result of histologic or clinical observations.

In typical manifestation, these atrophies are unilateral, slowly progressive, and ultimately are associated with the development of glaucoma.

Historical Note.—Whether G. Lindsay Johnson's (1) case of progressive atrophy of the iris, published in 1886, should be included in this group is uncertain. There is no statement as to the intra-ocular tension or visual field examination. Carl Hess's (2) record of a case of "non-traumatic iridodialysis," which appeared in 1892, is accepted by Clemens Harms, (3) who furnished in 1903 the first satisfactory illustrated clinical article on this subject. Other important Continental contributions, also illustrated, are those by Sven W. Larsson (4) (1920), Andor Licskó (5) (1923), Gottfried Arnold (6) (1923), and G. F. Rochat and W. Mulder (7) (1924).

The first reference to this subject in American literature is

ILLUSTRATING DR. GEORGE E. DE SCHWEINITZ'S ARTICLE, "ESSENTIAL PROGRESSIVE ATROPHY OF THE IRIS; A SECOND COMMUNICATION."

Plate 1.—Essential atrophy of the iris, May 25, 1926.

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by A. N. Murray, (8) who made a report for Casey A. Wood before the Chicago Ophthalmological Society in 1908. Two years later Wood (9) described this case in detail at the meeting of the American Academy of Ophthalmology and Oto-Laryngology. Other American contributors are William Zentmayer (10) (1913), G. E. de Schweinitz (11) (1915), Laura Lane (12) (1917), and Marcus Feingold (13) (1918).[1]

Pathologic investigations pertaining to this type of iris atrophy have been made by Bentzen and Leber, Casey Wood, Licskó, Rochat, and Feingold, the last named author's investigations being the most satisfactory and complete in this respect which have thus far appeared.[2]

Aberrations of the iris, characterized by holes or gap-formation, are well known; for instance, polycoria. Independently of those cases in which a "bridge-coloboma" of the iris exists, and of those where the normally situated pupil is crossed by a strand of persistent pupillary membrane in such a manner that two openings exist, are those types in which a number of openings are evident, variously placed and associated, it may be, with a large defect of the iris, in one direction.[3]

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[1] It is doubtful if Adolph Franck's case (14), (No. 7 in his list) should be admitted to this group. The patient, a woman of twenty-two, when examined, had simple glaucoma, the disc being typically cupped; the corneas were spotted with maculas, the result of "scrofulous keratitis" in childhood. Both irides exhibited a number of defects, partly complete and partly incomplete. By exclusion, the author concluded, although admitting that the history was deficient in this respect, that the iris anomalies were congenital in origin. Larsson, however, states that this case is in many respects analogous to the one he reports. Bentzen and Leber (15) examined microscopically an eye removed from a woman, aged twenty-seven years, in a state of absolute glaucoma. Below the widely dilated and fixed pupil, and separated from it by a small bridge of tissue, was "a second pupil," extending almost to the ciliary border. The only brief statement with respect to this "second pupil" is that in the patient's fourteenth year, near the pupil, a "black spot" was visible, which slowly increased in size (?). The question sign which the authors add to this record and the imperfection of the history obtainable would seem to exclude the case from the group of essential iris atrophies as defined in this paper. They attributed the patch of atrophy to the glaucoma.

[2] See Bibliography, Nos. 15, 9, 5, 7, 13.

[3] Referring to this variety of polycoria, Collins and Mayou (16) state: "There seems to have been in a large part of its circumference a failure

Such anomalies of the iris must not be mistaken for essential atrophy of the iris, ***which is not congenital***, and which is ***progressive***.

It is not improbable, however, that some of the reported cases of polycoria, associated with coloboma of the iris, were not congenital in origin, and really belong to the group which this essay attempts to describe. Indeed, one such case history, which I recorded in 1891 under the title "Coloboma of the Iris, Polycoria and Primary Glaucoma," (17) is strongly suggestive, in some of its aspects, of the group of iris atrophies now in consideration, and in the light of recent and more accurate knowledge, it is possible, even probable, that this lesion should have been regarded as one of the types of so-called progressive essential atrophy of the iris terminating in glaucoma—a condition, as we now understand it, with which I was not at that time acquainted. Doubtless other cases of this uncertain character are available. No attempt has been made to analyze the literature in this respect, especially as some reference to "polycoria cases" is made by Larsson in his comprehensive article (4). Doubtless, also an exhaustive review of the literature would reveal some case histories not referred to in this list, but those described are the important ones and suffice for the purposes of this essay. It is further evident that a number of typical progressive iris atrophies have been observed which have not been reported. Thus, I am aware of four such cases under the care of colleagues, who have described to me the iris lesions, but have not as yet recorded their studies of these patients.

AUTHOR'S CASE.—A brief summary of the case history reported in 1915 follows:

> The patient is an unmarried woman, aged twenty-three years (1915), whose parents are living and in good health. She is the third child in the family, and has two brothers and two sisters, living and well. She has had measles, scarlet fever, whooping cough, chicken-pox, pneumonia, pleurisy, and while attending school had adenitis of the

in the growth forwards of the secondary optic vesicle, from which the pigment epithelial layers of the iris are formed. The mesoblastic stroma, derived from the lamina irido-pupillaris, instead of forming a continuous layer, is deposited as bands between them."

neck and suppuration of the axillary glands; headaches were frequent. No areas of focal infection (teeth, tonsils, nasopharynx) were found. One year prior to the discovery of the iris lesions, the patient developed numbness, tingling, and loss of power in her right hand, of short duration.

Dr. D. J. McCarthy was called in consultation by her family physician, Dr. Council, and after a painstaking examination, including a negative Wassermann test and a thorough neurologic investigation, concluded that the nervous phenomena were functional, and that a latent tuberculous infection, causing a drag on the nervous system, was present, as well as a chronic intoxication from the gastro-intestinal tract.

The patient's eyes were examined by an oculist during her school days, and they were normal. On March 19, 1912, the late Dr. Frank Fisher found that the left iris (right eye normal) had the appearance of degeneration and thinning in spots, the pupil being egg-shaped and displaced toward the nasal side, with its point up and in. There was a small gap at the extreme outer margin near the base. Five months later the pupil had become more ovoid, and a second fenestra had formed above the first described. In January, 1913, there was an increase in the size of all the holes and in the width of the pupil. The fundus was normal. There is no mention in his report of the intra-ocular tension. Similar examinations and observations were made by Dr. John T. Carpenter at about this time.

The patient was first examined by me on July 9, 1913. Corrected vision was normal. The eyeground (left) contained no lesions; there was no rise of tension and the field was normal ($\frac{1}{300}$ white test-object).

The gradual development of iris atrophy, and displacement of the pupil to the nasal side from July 9, 1913, until May 5, 1915 (almost two years), are depicted in the accompanying diagram, reproduced from the colored plate published in 1915. (11) (Fig. 1, a, b, c.)

The first rise of intra-ocular tension occurred on April 30, 1914. This recurred from time to time, often accompanied by slight flushing of the eye and brow-pain, but minute examination failed to detect any evidence of iritis, or of the formation of posterior synechiæ. The "attacks" were temporarily controlled by myotics; operation was declined.

At the expiration of fourteen months the vision of the left eye was $\frac{6}{9}$ (the myopia had increased), two-thirds of the iris had completely disappeared, the gap being crossed by a strand of iris tissue, and the pupil was displaced to the

nasal edge of the cornea (Fig. 1, B). There was no cup, and no retinal vessel pulsation, but the tension was always slightly above normal. The media were clear, save only for a delicate haze in the corneal epithelium detected with a

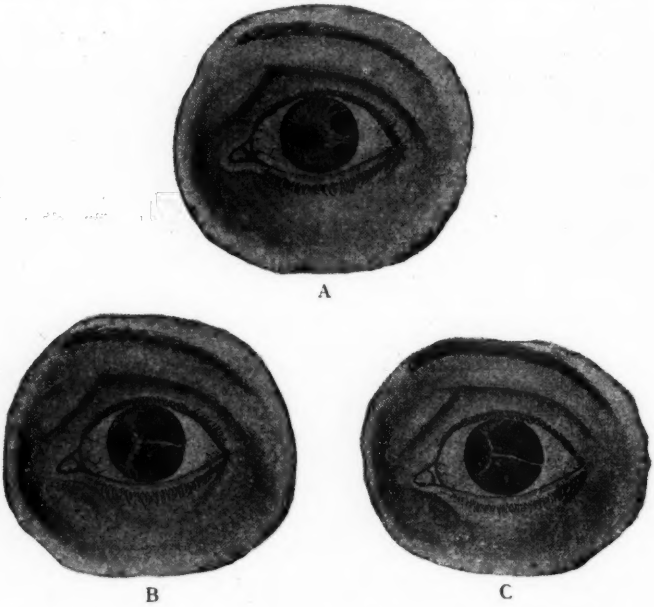

FIG. 1.—Essential progressive atrophy of the iris, showing gradual elaboration of the process.

loup. The right eye remained normal in all respects. The patient's general condition had not materially altered.

The history of this patient, after the report published in 1915, follows:

Clinical History from August 15, 1915, to Present Time.—After June 12, 1915, the patient was not seen until August 3, 1915, when she reported with the statement that she had experienced a number of visual obscurations. The ophthalmoscope revealed rapid venous pulsation; no cup; tension 35+ *mm* (Schiötz); V. = $\frac{6}{25}$, corrected to $\frac{6}{12}$; visual field

contracting, the depression being especially up and out (Fig. 2): the attack was controlled by myotics and dionin. Operation was suggested but declined.

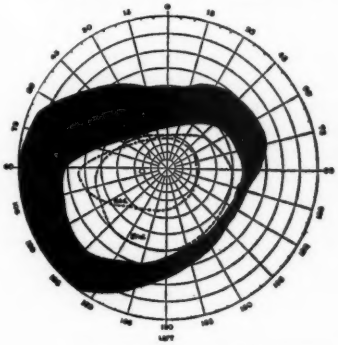

FIG. 2.—Left visual field obtained by test, $\frac{10}{1000}$, fourteen months after first rise of tension was noted (author's case).

FIG. 3.—Left visual field for white, $\frac{10}{1000}$, two years after Fig. 2 was charted (author's case).

During the following months, the patient being somewhat irregularly under observation, a number of similar attacks supervened, and on March 30, 1916, beginning cup formation in the disc was evident, and increasing myopia was present; the field of vision was contracted, especially in the upper and outer quadrant.

The same state of affairs continued during 1916, and at the expiration of one year, that is, on August 3, 1917, with -5.00 sph. $\bigcirc -1.00$ cyl. axis 150°, vision was $\frac{6}{12}$; the cup was deep and complete; the visual field was contracted, as shown in Fig. 3; and the tension was always above 30 (Schiotz), in spite of the vigorous use of myotics (operation was declined). The haze in the cornea, which was pronounced during the attacks, never entirely disappeared.

During the next year and a half, the cup in the disc deepened and widened; the field grew progressively smaller, and on January 30, 1919, presented the appearances depicted in Fig. 4, and vision was only $\frac{1}{60}$; tension very high, + + (not measured with tonometer); no additional changes in the iris.[1]

In August, 1920, although the cornea was hazy, the fun-

[1] All examinations of this patient in the period between April, 1917, and April, 1919, during the absence of the author, were made by Dr. T. B. Holloway or by Dr. B. F. Baer.

dus could still be seen, and the cup, very deep, was filled with new-formed vessels; tension + +.

Except once, the patient was not again seen until October 18, 1921, when she was attacked suddenly by pain in the *right*

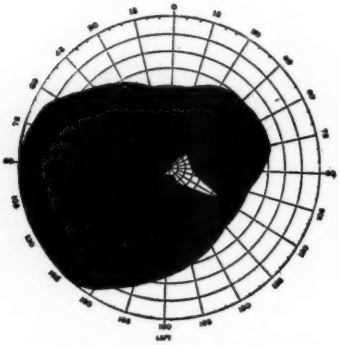

FIG. 4.—Left visual field for white, $\frac{10}{300}$, charted eighteen months after Fig. 3 was mapped; shortly afterwards blindness was complete.

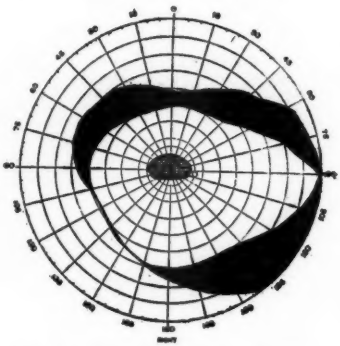

FIG. 5.—Field vision of and large central scotoma, *right* eye; retrobulbar neuritis (test-object $\frac{10}{300}$).

eye, especially severe on movement of the globe, and when it was pressed backward into the orbit; vision, counting fingers; no definite change in the disc; a large central scotoma in the center of the visual field—a typical acute retrobulbar neuritis. (Fig. 5.)

The patient was admitted to the University Hospital, and submitted to a thorough examination from all standpoints. Nothing of importance was elicited except as follows: Six years prior to this attack of retrobulbar neuritis, the patient had an attack of brachial neuritis, attributed to an abscessed tooth which was treated *in situ*, but not removed. Later, as the X-rays had detected an infected tooth, it was sterilized and filled. X-ray examination was now repeated, and a chronic abscess of the left upper first molar was discovered, and the tooth removed.

Examination of the paranasal sinuses yielded negative results; there were no abnormalities in the nasopharynx and larynx, but the faucial tonsils were infected. They were removed by Dr. Husik.

Rapid visual improvement followed, and at the expiration of two weeks the corrected vision of the *right* eye (+ .50 cyl. axis 150°) and the visual field were normal, and have remained so until the present time.

At the time the attack of right axial neuritis developed

no alteration took place in the *left* eye. Indeed, during the period from 1915 to 1921, except that the corneal haze had become too pronounced to permit a study of the fundus, no additional atrophy took place, and the iris did not materially differ from the appearances noted in Fig. 1, C.

During the evolution of the iris atrophy of the left eye, prior to the development of the corneal haze, it was not possible to study the iris tissue with the aid of a modern slit-lamp, although it was frequently investigated by means of a loup and the ordinary corneal microscope. The first slit-lamp inspection was made in May, 1925, by Mr. Basil Graves, and a little later by me, when, unfortunately, the corneal haze was pronounced The most evident features were a bullous and vacuolated condition of the corneal epithelium due to the high tension. This made detailed deeper examination impossible, so that we were unable to study the corneal endothelium, or the remnants of the iris tissue. (Plate I.)

The slit-lamp failed to reveal the slightest abnormality of the corneal endothelium or iris of the right eye.[1]

The most recent detailed ocular examination of this patient was made October 29, 1925. The results were as follows: Vision of right eye=$\frac{6}{6}$, accommodation ample, pupil reflexes normal. No fundus change except distinct pallor of the nerve-head. The visual field, however, obtained by test $\frac{10}{300}$, was normal in extent, and no central depression or scotoma was demonstrable, either for colors or white.

The condition of the left eye has been described (see preceding paragraph); T++ (tonometer not used); vision *nil.*

Because axial (retrobulbar) neuritis is not infrequently the forerunner of multiple sclerosis, Dr. D. J. McCarthy was requested to make a neurologic investigation. He submitted the following report: "Nothing was revealed by the examination on which a definite diagnosis of disseminated sclerosis could be based. The patient's speech is normal; there is no definite intention tremor.

"The reflexes of the lower extremities are exaggerated, but this exaggeration is not associated with any pathologic sole reflexes (Babinski, Gordon, etc.), or clonus. The abdominal reflexes are retained and active. There is no real loss of power, although the patient walks under examina-

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[1] Arnold also examined his patient's eye with the slit-lamp, and reports "some very fine point-like opacities in anterior cortex of the lens." There is no reference to any investigation of the iris or corneal endothelium by this method.

tion favoring the right leg. The cranial nerves, with the exception of those in relation to the eyes, are all normal.

"In 1913 she had an attack lasting three months that presented the picture of a functional (hysterical) hemiplegia affecting the right arm and leg. From this she completely recovered. In 1913, in addition to marked prostration, there was a loss of power again on the right side.

"Numbness of the right side (arm, leg and body) appeared in 1924, and for several days she could not walk. In January, 1925, she developed insomnia, imperfect balance when walking, loss of direction, and backache. At the present time (November, 1925) she is able to walk and be about normally, but is not strong."

Dr. McCarthy expressed the opinion that the motor and sensory phenomena in this patient were functional; *i.e.*, hysterical, but he calls attention to the persistence of the repeated "attacks" on the right side which might indicate an underlying beginning pathologic process, the nature of which is undiscovered.

Dr. McCarthy has continued his studies of this patient, and on March 11, 1926, wrote to me as follows: "The more I see of this case, the more I am inclined to your view of a potential disseminated sclerosis. The patient represents one of those extremely interesting cases where coming organic changes in the nervous system are foreshadowed by hysterical functional phenomena. This is a highly important subject, about which little had been written, and your patient represents a striking case with respect to it."[1]

COMMENT.

In the titular description of this group of iris atrophies, as previously defined, the words "spontaneous" and "primary" are sometimes included. Until our knowledge with respect to the etiolgic factors is more exact, they should be omitted. The term "essential," also used by several authors, is permissible as indicating the most conspicuous feature of the process.

The ages of the female members of the group (seven in number) noted at the time their clinical histories were recorded, were, respectively, 20, 23, 27, 28, 37, 41, and 47 years; those of the male members (five in number) were, respectively,

[1] The patient was last seen May 24, 1926. The conditions noted October 29, 1925, remain unchanged, except as portrayed in the drawing made at that time. (Plate I.)

14, 38, 43, and 44 years; one case not recorded. But the ages at which the iris affection was first noted were approximately: females, 18, 21, 26 (two cases), 20, not recorded, two cases; males, 8, 33, 40, and 43. The right iris was implicated in three cases; the left iris in eight cases; both irides in one case. There is some doubt, however, as previously has been mentioned, whether the last-named case, reported by Hess, should be included in this group.

The earliest sign of this affection with rare exceptions is an alteration of the normal disposition of the pupil, manifested either in its apparent enlargement, or, to use Rochat's words, in its "striking eccentricity."

This eccentric position of the pupil is well shown in the cuts which illustrate the case histories reported by Harms, Larsson, Lickso, Hess, Rochat, and myself.

The irregular enlargement of the pupil, due to the advancing atrophy, is indicated in the figures which illustrate Feingold's and Arnold's case records, and is reported by Casey Wood and by Laura Lane.

Rochat suggests that the eccentric position of the pupil may be "the result of a circumscribed shrinking of the iris by some pathological process," or "by assuming a primary atrophy of the iris, in which case the pupil would naturally be drawn to the side where the iris is less atrophic." This seems to explain what happened in the iris of my patient. The pupil, first almost horizontally elongated toward the nasal side, was gradually dragged by the less atrophic iris in the lower and inner quadrant into a vertical position, the advancing atrophy of the temporal side of the iris being unable to resist its efforts. A study of the other illustrations indicates that a similar mechanism may have been active in the displacement or the enlargement of the pupil.[1]

Most of the patients whose case histories are referred to in the text were studied when rise of tension, or glaucoma, had already supervened. But Casey Wood, in agreement with Harms, records his belief that it is quite possible, if they could

[1] From his anatomic examinations, Rochat concludes that the principal factor in the process is the soldering together of the root of the iris and the periphery of the cornea, beginning at a circumscribed spot and leading to displacement of the pupil.

have seen their patients early enough, before the iritic atrophy had much advanced, that the eyes *might* have been found without glaucomatous signs, as the history of Rochat's and my patient demonstrates.

Indeed, iris atrophy may be considerably advanced prior to the rise of intra-ocular tension, or the appearance of early glaucoma. My patient was under accurate observation for two years before any sign of glaucoma appeared, and during this period there was a steady increase in the atrophic process. Rochat had a similar experience, and, unacquainted with my publication, which antedated his by nine years, believed that his case was the first one in which a complete examination for glaucoma was made at an early stage.[1]

A gradually developing iris atrophy with hole formation had existed for two years before Arnold examined his patient, and yet there was no glaucoma.[2]

From these clinical data and others which might be adduced, it is evident that the iris atrophy antedates the establishment of glaucoma, and may maintain this order of precedence for a considerable period of time.

As previously stated, an early phenomenon of this affection, and one, indeed, which may first call attention to it, is an alteration in the disposition of the pupil. But, as Rochat points out, the pupil, even when a considerable portion of the iris has disappeared, remains intact, although usually displaced toward the periphery. This is particularly evident in the iris of my own patient, and also in the illustrations in the articles by Harms, Larsson, Hess, and Rochat.

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[1] Rochat and Mulder's patient was examined May 25, 1917, when iris atrophy was well-marked, but the eye was entirely free from any sign of glaucoma. It is expressly stated: "V. $\frac{6}{6}$; tension normal; no restriction of field of vision; fundus normal." How soon after this date the first signs of glaucoma appeared is not recorded. Six years later, when the patient was again seen, the atrophy of the iris was elaborate, the eye blind, and in a state of advanced glaucoma.

[2] For two years prior to Arnold's investigations of his patient, she had been under the care of two physicians who noted the beginning of the atrophic areas, and when Arnold saw her the process was advanced, but the eye, save for the iris lesion, was normal. Arnold appears to have examined his patient only twice (May 17, 1923, and July 31, 1923). Later history is not recorded.

Even where the atrophy is so disposed that a gradual irregular enlargement of the pupil, with especial peripheral displacement, interprets it, the outlying iris districts may exhibit large gaps or holes. Wood writes (the patient having been under observation from September, 1907, to the beginning of 1909): "fully five-sixths of the iris tissue had disappeared, and the pupil had assumed the shape of an irregular triangle, whose angles reached almost to the root of the iris, in which situation *all the layers of the iris* have become absorbed. Wood's examination of this iris with the corneal microscope indicated to him that "absorption of the iris tissue seems to have begun on the anterior surface, and to have proceeded irregularly, while the pigment layer has undergone absorption, chiefly, or only, at the pupillary edge.[1]

Rochat, referring to the anatomic examinations made by Bentzen and Leber, by Casey Wood, and by Licskó, writes: "Their results are mainly the same. . . . No signs of any inflammatory disease were found." In so far as Wood's investigations are concerned, this statement is not entirely accurate, inasmuch as his pathologist (Dr. Francis Lane) found lesions which he attributed to subacute and chronic fibrinoplastic iridocyclitis.

All pathologic investigations, including the recent ones of Rochat and Feingold, all the eyes having been in a state of advanced glaucoma, showed more or less obliteration of the angle of the anterior chamber, but Licskó remarks: "Die Iriswurzel liegt fest der Kornea an jedoch ohne Spuren einer Verwachsung," and Feingold, although he found "the iris root adherent to the cornea . . . in this way obliterating the angle of the anterior chamber," stated: "the case furnishes no explanation for the mechanism of the glaucoma, which is present in all these cases."

Rochat attributes the glaucoma which ultimately destroys

[1] Gottfried Arnold does not include Casey Wood's case in his collection of progressive iris atrophies, because, so he states, in the iris of Wood's patient there was "no hole formation, only atrophy of the anterior layers." It is true the "hole formation" is not described, but the loss of iris tissue was, in the periphery, complete, all of the layers having been absorbed.

vision to "the slowly progressing obliteration of the angle of the anterior chamber," and Licskó to dissemination of pigment from the atrophying iris (Koeppe's theory), and also to the diminished surface of the iris available for resorption of the intra-ocular fluid on account of iris atrophy (Hamburger's theory). Both C. Hess and Casey Wood regard the glaucoma as a secondary one caused by a slow or low-grade iritis. The latter author noted migration of the uveal pigment from the iris toward the sclero-iridian angle, where it could not escape on account of obliteration of the filtration area, due, he believes, to an exudative plastic iritis.

In none of the eyes included in this group were posterior synechiæ demonstrable, either by clinical or microscopic methods of examination. Clinically, no other signs of present or past iritis were found in any of the eyes belonging to the members of this group, except those observed by Hess, who found numerous white points (spots of exudate?) on the anterior capsule of the lens, and by Harms, who noted fine brown precipitates on the posterior surface of the cornea.[1] Anatomic investigation of the anterior part of the uveal tract of Wood's patient, as before stated, indicated the presence of fibrinoplastic iritis, although it is evident from the record that the lesions were in no sense elaborate. According to Feingold, the histologic evidences of any inflammation which might have caused the changes in the iris were even less marked than in Wood's case, and were limited to slight infiltration and pigmentation around Schlemm's canal.

It is safe to assume that even where a low grade of inflammation is evident in the iris tissue, it is not the true cause of the extensive atrophy which slowly supervenes. Moreover, it does not always exist, as Licskó's investigations prove.

According to the histories of the cases, injury in the ordinary sense of that term may be excluded as a causative factor.

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[1] Harms's patient, an iron-worker by trade, for more than twenty years was much exposed to the heat and glare of a smelting furnace. The pathologic effects of radiant energy on the crystalline lens are well known (glass-worker's, "puddler's," gold-smelter's cataract), and Harms thinks it possible, although improbable, that in these circumstances the iris suffered and atrophy ensued, and the pigment grains which were found on the posterior surface of the cornea were due to it, and not to a low-grade iritis.

From the standpoint of constitutional disturbance and infections, the following data are available: Scarlet fever eight years prior to examination (Hess's case); severe sciatica eight to nine years before investigation (Harms's case); purulent maxillary sinusitis, attributed to influenza, which, however, occurred about four years *after* the discovery of the iris atrophy (Wood's case); poor vision attributed to childbirth twenty-one years before iris atrophy was noted, but history uncertain (Larsson's case); syphilis, 4+Wassermann, but date of infection not given (Licskó's case); "light degree of struma" (Rochat and Mulder's case); acne vulgaris and probably latent tuberculosis (Lane's case); measles in childhood (Feingold's case); degraded nervous control dependent upon latent tuberculous infection; gastro-intestinal intoxication; potentially possibly a case of disseminated sclerosis (the author's case).

The long period of time during which my patient has been under observation, practically fourteen years, afforded unusual opportunities to study the conditions from all standpoints, which was carefully accepted, and the results detailed in the clinical history, and condensed in a sentence which appears in a previous paragraph.[1]

The attack of retrobulbar neuritis of the right eye two years ago, due to a tonsil and tooth infection, can hardly be related to the iris lesions, although it may be the forerunner of disseminated sclerosis, nor have any additional constitutional conditions developed which should be regarded as etiologically active.

In summary, therefore, it is evident that in typical cases of this affection the iris atrophy is not caused by the glaucoma, which is the end-result. Indeed, as the atrophy proceeds, the root of the iris is welded to the periphery of the cornea, a dissemination of pigment occurs, the iris surface available for resorption of the intraocular fluid diminishes, and glaucoma results. In a word, certainly in characteristic instances of this malady, the atrophic process originates the glaucoma, and not the glaucoma the iris atrophy.

Admitting the presence of a low-grade uveitis, apparently

[1] Licskó's patient was under observation for fifteen years.

present in two cases and doubtfully in a third, this of itself cannot be accepted as the true causative factor, because elaborate iridocyclitis, while it may create iris atrophy, never, so far as I am aware, creates such extensive destruction of the iris layers, and was, moreover, absent in a majority of the cases, and definitely proved to be so in two anatomic examinations.

The constitutional and local and general conditions which have been summarized from the clinical histories afford no satisfying evidence that they can be considered as definite causative agencies. Perhaps they may exert an influence which renders the iris tissue liable to the true cause, as yet undiscovered.

Naturally, speculations and theories are not lacking in this respect. Thus, Zentmayer suggested as an explanation of the condition a sclerosis of the vessels of the iris which creates an atrophy of the stroma from loss of nutrition, but does not commit himself to any theory as the cause of this vessel sclerosis.

Anatomic investigations have revealed some thickening of the vessel walls. Feingold, although he stated that his case offered no positive explanation of the atrophy of the iris, thought that some of the features of the histologic picture indicated a congenital vascular disturbance in the neighborhood of the smaller iris circle, which might have induced the change in the iris.

We know that pressure upon the vessels of the major circle of the iris may induce atrophy of its structure, hence a vessel disease or disturbance, impeding nutrition, may have a similar effect.

It occurred to me, as I pointed out in my first paper on this subject, that it is conceivable that a toxin liberated by some constitutional or local infection may be responsible for exciting "iris-vascular disease," whereby nutrition is lowered and atrophy follows. Acceptance of this statement, however, would require a favorable reception of the theory that such a toxin possessed a selective action in relation to iris-vessel tissue.

It was also suggested that the process might be described as neurotrophic atrophy, such as occurs in the skin in the

presence of neuritis (induced by a toxin, as previously referred to), and pressure on the supplying nerve plexus, something akin, only much more elaborate, to the marginal iris atrophies in tabetics, which Dupuy-Dutemps (18) attributes to compression of the ciliary nerve.[1]

Finally, one is tempted to turn to Sir William Gowers's theory of "abiotrophy," or degeneration of tissue due to defective vitality, in an endeavor to explain the origin and development of essential progressive atrophy of the iris. We are familiar with Treacher Collins's (19) interesting utilization of this theory in his discussion of the etiologic factors which pertain to a number of ocular disorders; for instance, certain types of hereditary postnatal cataract, hereditary ocular palsies, pigmentary degeneration of the retina, Leber's atrophy, and familial choroiditis. E. Fuchs, cited by Collins, has reported certain cases of bilateral ptosis, some being hereditary, which he ascribed, as the result of a microscopic examination of excised tissue, to a primary atrophy of the muscles, inasmuch as he found thinned fibers, increase in the number of nuclei, and pigmentary degeneration inside the sarcolemma.

In these cases, as well as in those types of chronic progressive bilateral paralysis of all the orbital striated muscles, described by Wilbrand and Saenger as a clinical entity, and which, according to Collins's belief, are due to a primary degeneration in the muscle fibers, the iris tissue invariably escapes. But this fact would not of itself preclude the possibility that essential progressive iris atrophy may be a form of abiosis.

"Ocular abiotrophies" are familial affections, although sporadic cases have been observed. There is no instance on record, so far as I am aware, of hereditary iris atrophy, that is, of the type with which this essay is concerned. If it is necessary to assume that an ocular abiotrophy must be hereditary, or must belong to familial affections, this assumption would put out of court the theory which has been advanced. However, Gowers has pointed out that "Similar cases occur in sporadic form, isolated, as do other family

[1] In the discussion which followed the reading of this paper, Calhoun suggested trophic disturbance due to lesion of the cervical sympathetic as a possible explanation of this condition.

maladies." Nevertheless, in spite of observations, clinical and anatomic, of speculations and theories, it must be admitted that the true cause of essential progressive atrophy of the iris and the formation of holes in its tissue, which may be regarded as representing a clinical entity, has not been discovered.

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AN EFFECTIVE TREATMENT FOR SYMPATHETIC UVEITIS.[1]

By Dr. F. H. VERHOEFF, Boston.

(From the Massachusetts Eye and Ear Infirmary, Boston.)

IT is generally admitted that the treatment of sympathetic uveitis has hitherto been highly unsatisfactory if not, aside from the use of atropine, entirely valueless. In cases in which the inflammation is severe soon after the onset the ultimate result is almost always complete or nearly complete blindness. Mild cases occur, the serous type, in which the eye shows little or no congestion or impairment of vision at any time and in which complete recovery takes place, but these are rare and even in such cases it is improbable that the course of the disease has been influenced by treatment.

The treatment that I now employ has apparently given remarkable results. The cases, however, in which I have had opportunity to use it have been relatively few. It consists in frequent subcutaneous or intramuscular injections of diphtheria antitoxin in large doses. The considerations that led me to employ it were, first, that horses were not subject to sympathetic uveitis and hence their serum might be antagonistic to the disease, second, that the serum of a horse immunized to diphtheria toxin probably contained enhanced non-specific factors of immunity; third, that it was remotely possible that the antitoxin itself might be antagonistic to the disease; fourth, that the serum might act as an antianaphylactic in case the anaphylactic theory of the disease was true.

Following are all the cases of sympathetic uveitis, seven in

[1] Read before the American Ophthalmological Society, Hot Springs, Va., June, 1926.

number, in which the treatment has been carried out under my direction. A case occurring at the same time as Case 1 is not included, because, although antitoxin was given, the injections were begun late and discontinued after the third on account of the occurrence of wide spread erythema and urticaria. Sight was ultimately lost. Subsequent cases have proved that anaphylactic symptoms do not contraindicate the continuance of the treatment. Case 2, was referred to me by Dr. Harry Goodall, Case 3, by Dr. Allen Greenwood, Case 4, by Dr. Joseph Dowling, and Case 5, by Dr. J. H. Waite. The diphtheria antitoxin employed was supplied by the Massachusetts State Board of Health. This is a concentrated antitoxic horse plasma from which the proteins other than the antitoxic pseudoglobulins have been largely removed. Twenty thousand units of antitoxin are contained in about 15*cc.* of the fluid.

CASE 1.—Albert C., aged 10. Admitted to Massachusetts Eye and Ear Infirmary June 6, 1921. On June 6, 1921, the patient had sustained a perforating wound of the cornea with prolapse of iris, right eye. June 7, the prolapsed iris was excised.

July 1. Enucleation right eye.

July 7. Slight congestion, left eye.

July 9. Congestion greater, definite Descemetitis and pigment dots on lens, V.O.S. = $\frac{20}{30}$.

July 11. Subcutaneous injection $\frac{1}{8}$ minim Coley's toxins. Temp. 99°, leucocyte count, 13,000.

July 13. Eye redder, exudate in pupil. Coley's toxins $\frac{1}{4}$ minim. Temp. 100°, leucocyte count, 13,800. Mercurial inunctions daily.

July 15. Coley's toxins $\frac{1}{2}$ minim. Temp. 100°, leucocyte count, 17,200.

July 18. V.O.S. = $\frac{20}{70}$. Descemetitis more marked. Temp. 99°, 5,000 units diphtheria antitoxin.

July 20. Temp. 100°, leucocyte count, 15000.5000 units antitoxin.

July 22. Temp. 99°, leucocyte count, 18400.5000 units antitoxin.

July 24. Marked erythema and urticaria. Mercurial inunctions discontinued.

July 27. Less congestion and Descemetitis. V.O.S. = $\frac{20}{60}$ with glass.

July 29. Improving. V.O.S. with + 1.75 S = $\frac{20}{40}$ −1.

Aug. 1. More congestion. Small nodule on iris. Sodium Salicylate.

Aug. 3. Considerable amount of fibrin in anterior chamber. Patient has follicular tonsillitis.

Aug. 4. Less congestion.

Aug. 8. Less congestion, but iris is swollen. V.O.S. = $\frac{2}{10}$ with glass.

Aug. 12. Tonsils and adenoids removed under ether anesthesia.

Aug. 16. Salicylates discontinued.

Aug. 18. No congestion. Descemetitis still marked. V.O.S. $\frac{2}{10}$ with glass.

Aug. 25. No congestion. V.O.S. with -3 S = $\frac{2}{10}$ -2.

Sept. 19. No congestion. Iris tied down. Delicate membrane over pupil. V.O.S. $\frac{2}{10}$ with glass. Discharged.

Dec. 29, 1921. Readmitted. Tension 28*mm* (Schiotz). V.O.S. = $\frac{2}{10}$ with glass. Pilocarpin 1% t.i.d.

Jan. 12, 1922. Tension 15*mm*. V.O.S. with -5.50 S = -1 cyl. ax. 150° = $\frac{2}{10}$ -1. Discharged.

May 14, 1923. Readmitted. Marked band keratitis. Secondary glaucoma. No congestion. V.O.S. = $\frac{2}{10}$ with glass.

May 15. Iridectomy. Iris could not be removed from lens but communication with posterior chamber was established above.

May 21. Tension normal. V.O.S. = $\frac{2}{10}$ with glass. Discharged.

June 16, 1923. Tension 30*mm*. V.O.S. = $\frac{2}{60}$. Cyclectomy.

June 27. Eye very soft. V.O.S. = $\frac{20}{200}$. Discharged.

Aug. 30. V.O.S. $\frac{2}{10}$ -2 with glass. Intradermal test with uveal pigment supplied by Dr. Allen Woods, negative.

May 7, 1924. Readmitted. Tension 32*mm* (Souter). V.O.S. = $\frac{2}{10}$ -2.

May 10. Subconjunctival incision of sclerotomy wound.

May 12. Tension 1.9*mm*. Discharged.

Sept. 11, 1925. Tension 20*mm* (Souter). V.O.S. with -6 S = -1.50 cyl. ax. 180° = $\frac{2}{10}$ -1.

Microscopic examination (4193) of the right eye, removed July 1, 1921 shows typical but early sympathogenic uveitis, with foci of epitheloid cells.

Case 2.—Helen A., aged 30. Admitted to private ward Massachusetts Eye and Ear Infirmary April 14, 1924, under the care of Dr. E. B. Goodall. At the age of 6, left eye was cut with a piece of glass. Eye blind ever since. Feb. 26, 1924, left eye was struck by a dog's head. Patient was seen in O.P.D. where enucleation was advised, but refused.

O.S. Greatly congested, shows old perforating wound of cornea extending into limbus, old band keratitis, and recent rupture of limbus. Eye full of blood, tension minus. Vision, nil. O.D. Normal, V.O.D. = $\frac{20}{20}$.

April 15, 1924. Ether. Enucleation O.S. with implantation of glass ball.

April 18. Discharged.

May 15. Patient complains that vision of right eye is slightly blurred. Very slight congestion. With ophthalmoscope a few deposits are seen on back of cornea. Fundus normal. V.O.D. = $\frac{20}{20}$.

May 7. Sodium salicylate by mouth, in increasing doses.

May 15. Condition progressing. Sodium salicylate, grs. 20, to be given daily intravenously.

June 5. Readmitted. Iris bound down, considerable amount of exudate in pupil. Marked congestion. V.O.S. fingers at 10 feet. 14 injections of sodium salicylate have been given. Discontinued. Saturated solution potassium iodide up to 20 drops, t.i.d. to be given.

June 20. Discharged.

June 21. No improvement. 15,000 units antitoxin. Fairly marked reaction at site of injection.

June 23. 15,000 units antitoxin.

June 25. 20,000 units antitoxin. General urticaria.

June 25 to July 28. Injections 20,000 units antitoxin every other day.

July 28. Readmitted. Very slight congestion. Iris completely adherent to lens. Pupil covered with membrane. Anterior chamber shallow. Tension 30*mm* (Souter). V.-O.D. = $\frac{30}{100}$.

July 29. Iridectomy by Dr. Verhoeff. Iris torn away from lens, leaving membrane behind.

July 30. Antitoxin 20,000 units.

Aug. 6. Tension remains high. Antitoxin 20,000 units.

Aug. 7. Iridectomy by Dr. Verhoeff.

Sept. 7. Eye white and quiet. Tension normal. Pupil and coloboma almost completely obstructed by membrane. V.O.D. = fingers at 1 foot. Discharged.

March 22, 1925. Readmitted. Eye has remained white and quiet. *Operation.* Linear extraction of lens by Dr. Goodall. Considerable amount of lens matter left.

April 4. Very slight reaction. Discharged.

April 27. Readmitted. Pupil and coloboma blocked by membrane. No congestion.

April 28. Operation. Discission of membrane. Good opening obtained.

May 2. No reaction. Discharged.

Nov. 30, 1925. Eye white and quiet. Tension normal.

Optic disc shows moderate glaucomatous cup. Field moderately contracted on nasal side. Vision with cataract glass = $\frac{20}{40}+$.

April 21, 1926. Vision with cataract glass = $\frac{20}{20}$.

Microscopic examination (5218) of left eye removed April 15, 1924, shows old perforating wound of corneal limbus, traumatic cataract largely absorbed, band keratitis, old iridocyclitis. More recent rupture of wound with exposure of ciliary body. Sub-choroidal hemorrhage. Recent sympathogenic uveitis with foci of epithelioid cells.

CASE 3.—Nettie M., aged 40. Admitted to Massachusetts Eye and Ear Infirmary May 17, 1924. About two years ago she developed glaucoma in left eye and soon afterwards iridectomy was done upon it by Dr. Greenwood. Two months ago, the tension still being high, he performed iridotasis. Three weeks ago the left eye became inflamed and the vision blurred. The vision of the right eye has always been very poor—congenital amblyopia. Dr. Greenwood made a diagnosis of sympathetic uveitis and referred the patient to this hospital with the request that the patient be treated with intravenous injections of sodium salicylate as recommended for such cases by Melville Black. On examination both eyes show congestion and marked Descemetitis. V.O.D. = $\frac{3}{200}$, V.O.S. = light perception faulty.

May 22. No improvement. Sodium salicylate has been given intravenously daily in doses of 20 grains. Discontinued.

June 9. Daily injections of sodium salicylate resumed.

June 21. No improvement. V.O.D. = fingers at 1 foot. V.O.S. = projection faulty. Discharged at request of patient.

July 2, 1924. Readmitted. O.D. Marked congestion, marked coarse Descemetitis. Iris bombe. Pupil Semidilated, filled with exudate. Tension elevated. O.S. Marked congestion. Exudate in pupil. Tension normal. Leucocyte count 9,200. Subcutaneous injection 20,000 units diphtheria antitoxin given. To be repeated daily.

July 3. Leucocyte count, 16,400.

July 7. Marked improvement, both eyes. Congestion less, iris less swollen.

July 9. Left eye nearly white. Slight urticaria.

July 15. Iridectomy O.D. Profuse hemorrhage.

July 19. Blood in anterior chamber absorbing.

July 25. Blood absorbed. V.O.D. fingers at 2½ feet.

July 26. Antitoxin discontinued.

Aug. 4. Both eyes white and quiet. V.O.D. = fingers at 2 feet. Discharged.

Sept. 26. Readmitted. Eyes remained quiet until 2

weeks ago when O.S. became painful. O.D. White. Descemetitis. Posterior synechia. Pupillary membrane. Tension normal. O.S. Congested. Descemetitis. Tension low. Test for sensitiveness to antitoxin negative. Subcutaneous injection 20,000 units antitoxin.

Sept. 28, 30, Oct. 2, 5, 6, 8, 10, 12, 20,000 units antitoxin.

Oct. 3. Wassermann negative.

Oct. 14. Symptoms of anaphylaxis-asthma, nausea, rapid weak pulse. V.O.D. – shadows. V.O.S. – projection faulty.

Oct. 6, 1924. Both eyes free from congestion, but Descemetitis persists. Discharged.

May 18, 1926. Patient has not visited the clinic since date of previous note, but states that both eyes remained free from irritation. Both eyes are now free from congestion. Corneæ clear, no Descemetitis. Right eye, tension minus, vision nil. Left eye, tension slightly minus. Anterior chamber normal depth. Pupil and coloboma blocked by membrane. Vision, light perception with perfect projection. 20,000 units antitoxin given. Operation left eye: keratome incision. Large piece of iris with pupillary membrane excised with Noyes scissors. Lens removed. Free irrigation.

May 19. Anterior chamber reformed. Marked striped keratitis.

May 26. Very slight congestion. Striped keratitis much less. Large clear pupillary opening. Tension very low. Vision handmovements.

Microscopic examination (5294) of the piece of iris obtained from right eye by iridectomy on July 15, shows typical picture of sympathetic uveitis, with foci of epithelioid cells.

Case 4.—Ernest L., aged 38, referred by Dr. Joseph Dowling and admitted to private ward Massachusetts Eye and Ear Infirmary, January 6, 1925, under the care of Dr. Verhoeff. Left eye was injured 5 weeks previously, a large foreign body passing entirely through it and lodging deep in the orbit as shown by X-ray examination. The eye was removed twenty-three days after the injury. On January 6 there is marked ciliary congestion, the iris is congested, pupil small. Under atropine the pupil dilates only in places, showing numerous posterior synechiæ. The anterior surface of the lens is dotted with pigment. Daily subcutaneous injections of 20,000 antitoxin begun. Sodium salicylate gr. *X* t.i.d. 1% atropine instillations every two hours.

Jan. 7. Pupil semi-dilated, some of the synechiæ giving way. Temp. 97°.

Jan. 9. All synechiæ have given away. Antitoxin given intravenously today. Temp. 99°.

Jan. 10. Still considerable congestion. Temp. 99°.

Jan. 12. Much less congestion. Temp. 102°, pulse 110 in afternoon.

Jan. 13. Eye almost free from congestion. Vitreous clear. Surface of lens dotted with fine pigment. Pupil dilated *ad maximum*. Marked hyperemia of buttocks about site of injections. Temp. 98°. Antitoxins discontinued. Patient has received seven injections to date.

Jan. 14. No congestion. V.O.D. = $\frac{1}{10}$. Slit lamp shows aqueous loaded with pigment, surface of lens coated with pigment granules, delicate striæ but no deposits on back of cornea. Patient discharged.

Nov. 10, 1925. Dr. Dowling reports that the eye has remained quiet and is normal except for a circle of pigment left on the lens by the synechiæ. The vision is normal.

CASE 5.—The history of this case is too long to be given in complete detail. The patient, a large man aged 38, was admitted to the private ward of the Massachusetts Eye and Ear Infirmary February 13, 1925, under the care of Dr. J. H. Waite. On Feb. 4, 1925, his right eye had sustained a perforating wound of the cornea and ciliary body with injury to the lens. On Feb. 13, the prolapsed iris and ciliary body were excised and the wound covered with a conjunctival flap. The congestion of the eye gradually subsided somewhat but in about six weeks began to increase again. On March 28, the left eye was still perfectly normal on slit lamp examination, and had vision = $\frac{20}{15}+$. On April 4, the right eye showed faulty projection and the left eye Descemetitis. The left eye was enucleated and treatment with antitoxin begun, 20,000 units being given daily, until April 17, when marked serum sickness developed —skin rash, multiple arthritis, temperature 103°, nausea and vomiting. On the second day after beginning the treatment the eye was considerably congested and there were several fine posterior synechiæ. The congestion then increased somewhat, but on the tenth day was almost gone. The antitoxin treatment was discontinued for about three weeks, during which time the ocular inflammation became active again and the vision reduced to $\frac{20}{200}$. May 11, 20,000 units of antitoxin were given in divided doses. Two days later the eye showed marked improvement. May 20, 20,000 units were given and on May 23, V.O.S. = $\frac{20}{15}$. All congestion disappeared and on July 11, V.O.S. = $\frac{20}{15}-2$. Nov. 14, the eye was still white and quiet and the vision $\frac{20}{15}$. Nov. 16, there was a recurrence—congestion of eye,

haziness of media, and vision reduced to $\frac{2}{4\frac{0}{0}}$. 20,000 units were given in divided doses and within 48 hours the vision was $\frac{2}{2\frac{0}{0}}$. Nov. 27, V.O.S. = $\frac{2}{2\frac{0}{0}}$. At this time the pupil was widely dilated but the iris was apparently everywhere fused to the lens capsule. The anterior chamber was of normal depth, tension normal. Dec. 26, there was another recurrence with vision reduced to $\frac{2}{2\frac{0}{0}\frac{0}{0}}$, 30,000 units given. Dec. 28, V.O.S. = $\frac{2}{1\frac{0}{0}}$, 20,000 units given. Dec. 29, V.O.S. = $\frac{2}{2\frac{0}{0}}$. Jan. 15, 1926, Jan. 28, Feb. 10, Feb. 27, there were recurrences which subsided under antitoxin but, owing to the further development of pupillary membrane left vision reduced to $\frac{2}{4\frac{0}{0}}$ with glass. After this the lens began to show increasing cloudiness and on April 9 there was an attack of acute glaucoma with shallow anterior chamber and tension of 40*mm.* Paracentesis of the anterior chamber lowered the tension temporarily, but posterior scleral puncture reduced the tension to normal. Antitoxin was given and iridectomy then performed by Dr. Waite without causing any untoward reaction. May 8, 1926, the eye was white and quiet, and tension normal, but the field was greatly contracted as result of the glaucoma, and the vision reduced to counting fingers,—apparently by the cloudiness of the lens. The lens will later be removed and it is expected that useful vision will be obtained.

Microscopic examination (5895) of the right eye, removed April 4th, shows the typical picture of sympathogenic uveitis, with diffuse lymphocytic infiltration and numerous foci of epithelioid cells and giant cells. The whole uveal tract is markedly involved, but the process is most intense at the fundus of the eye where the choroid is increased to over three times its normal thickness.

Case 6.—Jennie G., aged 47. Admitted to Infirmary July 8, 1925. The right eye has been red and painful for 8 weeks. It shows marked congestion, anterior chamber very shallow, cornea hazy, pupil dilated. Tension 35*mm.* Projection faulty. Left eye shows very shallow anterior chamber but it is otherwise normal and its vision with + 2.25S = $\frac{2}{2\frac{0}{0}}$.

July 9. Iridectomy O.D. One pillar of coloboma allowed to remain in wound.

July 11. Tension normal. Discharged.

Aug. 4. Readmitted. O.D. Tension normal. Congestion at site of wound. Slight Descemetitis. O.S. Congested and painful. Subacute glaucoma. Tension 40*mm.* Pilocarpin 2% every three hours.

Aug. 6. O.S. white. No pain. Tension 10*mm.* V.O.S. = $\frac{2}{2\frac{0}{0}}$ with glass. Pilocarpin 1% t.i.d. Discharged.

Aug. 10. Readmitted. O.D. Ciliary congestion. Precipitates on lens. O.S. Ciliary congestion. Descemetitis. Precipitates on lens. Iris congested. Cells seen in aqueous with slit-lamp. One posterior synechia and some exudate in pupil. Tension 12*mm*. Fundus normal. V.O.S. = $\frac{10}{20}$ with glass.

Aug. 11. O.S. Pupil has dilated under atropine except at site of synechia.

Aug. 12. O.S. Adrenalin 1:100, $\frac{1}{3}$*cc*., subconjunctivally. 20,000 units antitoxin.

Aug. 13. Synechia still holds. 20,000 units antitoxin.

Aug. 14. Temp. 101°. 20,000 units antitoxin.

Aug. 17. O.S. Tension 34*mm*., cornea steamy. Desensitized, and 20,000 units antitoxin given.

Aug. 19. O.S. Tension 45*mm*. (Schiotz). Temp. 102°. *Iridectomy*. 20,000 units antitoxin.

Aug. 20. O.S. Anterior chamber reformed, contains much blood. Both arms red, swollen and tender from injections.

Aug. 23. O.S. Atropine 1% t.i.d.

Aug. 30. O.S. much improved.

Sept. 4. Both eyes white and quiet. V.O.S. with + 4S = +1 cyl. ax. 5° = $\frac{10}{10}$ −1. (The reduced vision O.S. is due to the iridectomy.) Discharged.

Sept. 11. In O.P.D. O.S. Tension normal. Very slight congestion (atropine?). 15,000 units antitoxin given as precautionary measure.

Sept. 8. O.S. Atropine conjunctivitis. Atropine discontinued.

Sept. 25. O.D. Congestion at site of wound only. Lens surface covered with pigment dots. O.S. White. Lens surface covered with pigment dots. Slit-lamp shows one typical nodule on pupillary margin and fine transparent deposits on back of cornea.

Oct. 30. Both eyes white and quiet. Tension O.D. 35*mm*., O.S. 10*mm*. (Souter). V.O.S. with +1.75S = $\frac{10}{10}$+.

Jan. 8, 1926. Unchanged.

Microscopic examination (6127) of the piece of iris obtained from the left eye by iridectomy on Aug. 19, was unsatisfactory owing to the traumatized condition of the specimen, and neither confirmed nor excluded the diagnosis of sympathetic uveitis. The stroma was almost free from infiltration but there seemed to be some foci of epithelioid cells beneath the pigment layer.

Case 7.—Tony G., aged 6, admitted to Massachusetts Eye and Ear Infirmary Nov. 10, 1925. Seven weeks ago while cutting a shoe lace, the knife slipped and cut the

left eye. Patient was treated by Dr. Merrill of Lawrence who advised removal of the eye and referred the patient to this hospital.

Examination: Left eye. There is a healed perforating wound of the cornea below which extends into the sclera. The iris is caught in the wound and the anterior chamber is obliterated. Very slight congestion. There is a good fundus reflex but no details can be made out with ophthalmoscope. The tension is very low. Vision, light perception with faulty projection. Right eye. Slight ciliary congestion. Pupil well dilated by atropine. Ophthalmoscope with +20D lens, shows numerous fine pigment dots on back of cornea and on anterior surface of lens. Vitreous apparently clear, fundus normal. Slit-lamp shows aqueous loaded with cells and many fine deposits on back of cornea.

Nov. 10. Operation, ether. Simple enucleation, left eye. 5000 units diphtheria antitoxin injected intramuscularly.

Nov. 11. Ciliary congestion gone. Leucocyte count 6,600. 7,000 units antitoxin given.

Nov. 12. Cornea still shows many pigment deposits. 6,000 units antitoxin.

Nov. 13. Deposits on cornea and lens less. Leucocyte count 6,000. Differential count, polynuclear 56%, eosinophiles 2%, small mononuclears 26%, large mononuclears 4%, transitionals 12%, hemoglobin 80%. 8,000 units antitoxin given.

Nov. 14. No deposits on cornea seen with ophthalmoscope. 8,000 units antitoxin given.

Nov. 15. Slit-lamp shows a few precipitates on back of cornea. Aqueous clear. 8,000 units antitoxin injected.

Nov. 16. 8,000 units antitoxin.

Nov. 17. No deposits on cornea or lens seen with ophthalmoscope. Two small pigment dots seen floating in aqueous.

Nov. 18. Eye apparently normal except for atropine mydriasis. No congestion. Vision $\frac{20}{20}-$. Discharged.

Dec. 11. Eye white and quiet. Three pigment dots visible on lens with ophthalmoscope, none on cornea. Slit-lamp shows a few pigment dots on back of cornea and a few cells in aqueous. V.O.D. = $\frac{20}{20}-$.

Microscopic examination (6255) of left eye shows typical picture of early sympathogenic uveitis with foci of epithelioid cells.

Before attempting to draw any conclusions as to the value of the treatment in these cases it is important to be as sure as possible of the diagnosis in each. In four cases this was

made certain by microscopic examination of the exciting eye and in Case 3 by microscopic examination of the iris tissue removed from the sympathizing eye. Of the other two cases, Cases 4 and 6, no material was available for microscopic examination in one, and the piece of iris excised in the other was unsuitable. The diagnosis in these two cases, therefore, rested entirely on clinical findings. In Case 4 the left eye became affected about five weeks after a severe perforating injury to the right and twelve days after removal of the latter. The onset was painless. The lens surface was covered with pigment dots, the iris was congested, and there were a number of posterior synechiæ which at first resisted atropine. There were considerable ciliary congestion, photophobia, and tenderness to pressure. Under the treatment the eye was practically normal within a week. In Case 6 both eyes painlessly developed Descemetitis, pigment deposits on the lens, posterior synechia, and ciliary congestion thirty-three days after an iridectomy upon the glaucomatous right eye with incarceration of the iris in the wound. Congestion about site of the wound had persisted. Both eyes showed prompt improvement under the treatment, so much so, in fact, that an iridectomy performed upon the left eye for increased tension caused almost no reaction. By means of the slit-lamp a nodule, such as claimed by Koeppe to be characteristic of sympathetic uveitis, tuberculosis, or tropical syphilis, was found on the pupillary margin of the left eye. The diagnosis in these two cases was, therefore, as certain as it possibly could be without microscopic examination.

As might have been expected recovery was most prompt and complete when the treatment was begun early as in Cases 4, 6, and 7. In four of the cases practically normal vision was finally restored. In Case 2, in which the treatment was begun late, the lens had to be removed to gain normal vision. In Case 1 vision of $\frac{20}{40}$ was obtained, but no doubt the final result would have been still more satisfactory if the treatment had been begun sooner and pushed more vigorously.

Case 3 is the only one in which the visual result was entirely unsatisfactory, but this was unquestionably on account of the long delay, nine weeks, before the antitoxin treatment was begun. The second eye was congenitally amblyopic and the

exciting eye was almost blind, having faulty projection, when the patient came under my care. Valuable time was lost in employing intravenous injections of sodium salicylate as recommended by Melville Black, for, as in Case 2, they were without the slightest benefit. This case nevertheless furnished strong evidence of the value of the antitoxin treatment, since the effect of the latter in relieving the ocular pain and congestion was most striking and left little doubt that useful vision could have been saved if the treatment had been instituted at the onset. When one considers the dense infiltration and injury to the uveal tract seen microscopically in advanced stages of the disease, it is obvious that it would be impossible to restore vision if the beginning of effective treatment was long delayed.

Clinically, Case 5 seemed to be an early one when the treatment was begun, but microscopic examination of the exciting eye removed at this time showed extremely marked involvement of the choroid thus indicating also marked involvement of the other eye. Under the treatment the vision improved in three months from less than $\frac{20}{200}$, to $\frac{10}{20}$ which vision was maintained for the next four months. After this there were a number of recurrences which subsided promptly under antitoxin but which caused an increase in the pupillary membrane and cloudiness of the lens. One year after beginning the treatment there was an attack of acute glaucoma which was relieved by scleral puncture and iridectomy. This left the visual field greatly contracted and vision reduced to counting of fingers. As far as could be determined the poor central vision was due to the pupillary membrane and cloudy lens, so that removal of the lens will probably restore useful vision. While this case demonstrated the effectiveness of the treatment in overcoming the original attack and each of the subsequent recurrences, it raises the question whether or not it is possible completely to eradicate the disease in extremely severe cases. We believe that the failure to do so in this case was due to the fact that owing to the development of severe anaphylaxis the treatment was discontinued at an early period for about three weeks. Judging by our further experience, if we had desensitized the patient at the first signs of anaphylaxis the serum sickness might have been

prevented and the antitoxin treatment continued without interruption. It is evident also, that the iridectomy should have been performed at an earlier period, but we hesitated to do this while the tension and vision were normal.

It is generally believed that it is disastrous to operate upon an eye that has been affected with sympathetic disease unless it has been quiet for at least one year, and this accords with my previous experience. Cases 2, 3, and 6, however, prove that if the patient is kept under the influence of antitoxin an iridectomy may safely be performed for increased tension early in the course of the disease, and Case 2 proves in addition that after the eye has been quiet for six months the lens may safely be removed.

In all except Case 7 symptoms of anaphylaxis occurred during the treatment. The development of anaphylaxis, however, was possibly not of essential importance, because improvement occurred before this became manifest. On the assumption that repeated slight anaphylactic reactions might be of benefit, after the patient in Case 5 became extremely hypersensitive small doses of antitoxin were injected daily, each producing a local reaction. No benefit resulted, but on desensitizing the patient and resuming the full dose of 20,000 units improvement promptly occurred.

In all except one case sodium salicylate was given along with the antitoxin. Case 5 in which the salicylate was discontinued while the eye was still in an active stage, and Case 7 in which it was not given at all, go to prove that it is not an essential adjunct to the antitoxin treatment. It is possible, however, that it may be of some benefit and it is therefore advisable to continue to use it, especially in cases in which there is congestion and pain.

Many questions arise in regard to this treatment that I am at present unable to answer. Would not simple horse serum or citrated plasma be equally as effective as diphtheria antitoxin? Would not other anti-serums serve as well? Would not equally good results be obtained if the injections were given at longer intervals? Would not unconcentrated antidiphtheric serum be preferable? To answer these questions it would be necessary to experiment upon patients who are in imminent danger of losing their sight and possibly to fail

to give them the benefit of effective treatment. The question as to whether or not larger doses of antitoxin would be preferable can no doubt be determined without jeopardizing the patients sight. The important question whether or not injections of antitoxin will act prophylacticly will no doubt be difficult, although not impossible to answer.

From my experience in the treatment of these cases I conclude that the following is the best procedure in cases of sympathetic uveitis. The exciting eye is removed only if it is so badly injured that there is no reasonable chance that it will recover useful vision. The patient is tested for hypersensitiveness to antitoxin, by injecting one drop intradermally. If there is no reaction within thirty minutes, the test is negative. If negative, 20,000 units of diphtheria antitoxin are heated to body temperature and injected subcutaneously or intramuscularly, preferably into the buttocks. If the test is positive, the patient is desensitized by injecting the antitoxin at intervals of 15 minutes in divided doses, .1*cc*, .2*cc*, .5*cc*, 1*cc*., 1.5*cc*., 2*cc*., 2.5*cc*., and then the remainder of the 20,000 units. In the case of children the dose is reduced in proportion to body weight. The same dose is given daily for one week. If the case is an early one and the ocular congestion has subsided within this time the injections are discontinued for one week and then given at weekly intervals for two or three weeks. If the case is an advanced one, the daily injections are continued until marked improvement has taken place or the patient has developed severe symptoms of anaphylaxis. They are then given at weekly intervals, each time in divided doses. In cases with marked congestion sodium salicylate is given in increasing doses but not to the limit of toleration. Locally, atropine is employed in the usual manner. If the pupil does not dilate fully, or if there is increase in tension, daily subconjunctival injections of adrenalin are given. If the tension remains over 28*mm* (Souter), iridectomy is done as soon as the eye otherwise shows marked improvement. If there is a pupillary membrane which reduces vision too greatly, and the eye has remained quiet for 3 months or longer, the lens is removed, the patient being kept under the influence of antitoxin by daily injections before and after operation.

LEPROSY OF THE EYE.

An Analysis of the Records of 512 Cases of Leprosy in the Hawaiian Islands.

By Dr. FORREST J. PINKERTON, Ophthalmologist to Kalihi Receiving Station, Consulting Ophthalmologist to the Board of Health, Honolulu, Hawaii.

(*With six illustrations on Text-Plate IV.*)

WHILE other workers are busy seeking a cure for leprosy, it seems to me that oculists should make every effort to save, even though temporarily, the sight of the victims of the disease. To this end I am presenting a résumé of my records in the examination of 512 cases of leprosy in Hawaii, ignoring, because of inadequate records, 90 other cases which I have examined.

363 cases were among the 518 lepers now isolated at Kalaupapa, Molokai, and the remaining 149 cases represent the total number of patients at Kalihi Receiving Hospital, Honolulu.

The 512 cases are classified racially as follows:

Pure Hawaiian	202	Korean	14
Caucasian	61	Filipino	31
Caucasian-Hawaiian	89	Porto-Rican	5
Japanese	53	Porto-Rican-Hawaiian	1
Japanese-Hawaiian	3	Fijian	1
Chinese	23	Negro-Hawaiian	1
Chinese-Hawaiian	27	Indian-Hawaiian	1

My notes are based upon a brief leprous history of each patient: upon my methods of treatment; and upon observa-

ILLUSTRATING ARTICLE OF DR. FORREST J. PINKERTON, ON "LEPROSY OF THE EYE. AN ANALYSIS OF THE RECORDS OF 512 CASES OF LEPROSY IN THE HAWAIIAN ISLANDS."

FIG. 1.—Neural type, natural amputations.

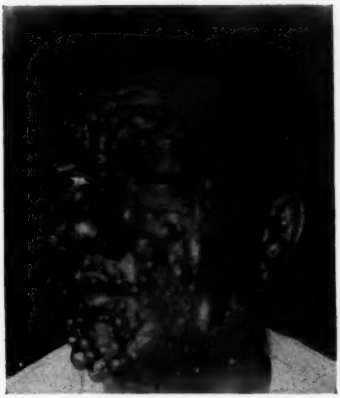

FIG. 2.—Nodular type, eyes entirely free of nodules.

FIG. 3.—Hypertrophy of the accessory lacrimal gland in a case of complete orbicular paralysis.

FIG. 4.—Orbicular paralysis complete. Forced effort to close the eyes. Neural type.

IG. 5.—Lepromatous granuloma, the end result of nodules at the limbus, invading the deep corneal tissue.

FIG. 6.—Nodule of the sclera at external limbus, early ribbon-like circumcorneal infiltration.

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tions made in examinations by bright daylight, by a powerful electric light, by the use of a strong magnifying glass, Berger's binocular loupe, and the electric ophthalmoscope. Since a further understanding of the leprous involvement of the cornea, the anterior chamber, and the iris, would probably be the greatest forward step in the treatment of these poor unfortunates, I have felt deeply the lack of a slit-lamp.

In the group of 363 cases at Kalaupapa there were 112 females, and 251 males. The average age was 37.3 years; the youngest patient aged 10, the oldest 74. 323 had some defect of the eye or adnexa, one or both. 40 (average age 26) were apparently unaffected, of whom 27 were neural, 10 mixed and 3 nodular cases of leprosy. 79 of the patients or 21.7% were blind in both eyes; 24 in the right eye, and 21 in the left. 61 eyes were blind as the result of atrophy of the bulb or of sclerosis of the cornea; 59 because of exudative iritis; and 81 because of lepromatous granulomata of the cornea. 138 or 38% had lid palsy or paralysis of the orbicularis with the usual ectropion, epiphora, and corneal changes. 141 or 39% had exudative iritis. 105 eyes had marked pannus formations, and 161 had leucomatous areas over part or all of the cornea.

Since the following data were not ascertained from 108 patients, I shall digest only 255 records henceforth. In this group there were 175 males and 80 females. Their average age at the first symptom of leprosy was 25; at first apprehension 27—the youngest having been 2 years old, the eldest 70. We may suppose, therefore, that there was an average of 2 years per patient of freedom, a menace to the community, from the time of his first symptom or leprous lesion, to the time of his apprehension and quarantine.

We classified the patients according to their first symptoms of leprosy as: neural 89 (Fig. 1); mixed 96 and nodular 70 (Fig. 2). We classified them as to types at the time of examination as: neural 87, mixed 145, and nodular 23. Since these patients were all at Kalaupapa it will be understood why they were all classed as advanced cases.

30 cases had refused treatment in any form, but 165 had had the ethylesters of chaulmoogra oil by intramuscular injections, 19 had taken the crude oil in capsule by mouth, and

41 had taken both the injections of the fatty acids and the crude oil by mouth. The average length of treatment by injections was 2.7 years, and by capsule medication 3 years.

16 patients had been paroled, one twice. The longest out on parole was 12 years; the shortest time was 1 year; an average of 5 years out on parole. In all probability these patients were active lepers and a distinct menace to the community for the greater part of that time.

Of the 255 patients, 89 gave an early history of pain in their eyes (probably iritis), 136 of redness (probably iritis or the usual causes) and 76 of lacrimation only (probably orbicular paralysis); 85 had reduced vision in both eyes (probably caused by corneal changes or exudative iritis); and 27 had reduced vision in the right eye, 25 in the left eye only.

At the time of examination 155 of these patients had facial fibrillation or tremor. In 58 cases the accessory lacrimal gland was hypertrophied. 38 had nodules on the lids, 41 had nodules on the globe, and 20 had nodules on the iris. In 62 cases the brows were totally absent, and in 50 cases both upper and lower lashes were totally absent. The brows were thinned or partially gone in 61 cases; the lashes in 78 cases—the lower lashes being thinner than the upper, and by far the first to entirely disappear.

The record of operative work and other treatment of the eyes which I have done at Kalaupapa, from June, 1924, to March, 1926, is as follows:

Peridectomy both eyes, 14, right 44, left 33.
Internal lid suture, both eyes 5, right 12, left 5.
External lid suture, both 8, right 34, left 23.
Iridectomy, right 10, left 17.
Enucleations, both 2, right 9, left 6.
Excision of lepromata, right 17, left 15.
Ptergyium resections, right 6, left 3.
Extractions of cataracts, right 1, left 4.
Needling operations, right 1, left 2.
Refractions 10.
Medical treatment 52.
Refused operative treatment 38.
Refused medical treatment 10.

It is interesting that a total of 301 operations, all under local anæsthesia, were performed without a single case of postoperative infection, despite the fact that working conditions were such that the technic of asepsis was not of the best. All we could do was to be decently clean, scrubbing the operative field with synol soap and water, and flushing the surface with boric or dilute bichloride solution. From the results, however, it appears that leprous patients are more immune than most to the ordinary infectious organisms.

In the group of patients at Kalihi we have a total of 149—99 males and 50 females. Their average age at the present time is 28; the youngest patient is 9, the oldest is 78. The average age at which symptoms first appeared was 23. The first symptoms appeared in one patient when but a babe in arms; in the oldest at the age of 67. The average age at apprehension was 24.3 years; the oldest having been 69, the youngest 8. We classified them as to type from their first symptoms as: neural 52, mixed 68 and nodular 29.

36 patients had been paroled. Think of it! 23½ per cent. of the patients at Kalihi are returned parolers. They have been free to roam the country an average of 3½ years each. 4 of these returned parolers had been given an absolute release from parole by Medical Boards made up of local physicians and after being at large an average of 7 years each, were reapprehended as advanced or moderately advanced cases. The group as a whole was classified upon readmission as follows: Neural moderately advanced 3; neural advanced 6; mixed moderately advanced 16; mixed advanced 5; nodular moderately advanced 3; and nodular advanced 2.

Of the 149 lepers at Kalihi, 110 had in greater or lesser degree some lesion or involvement of the lids, globe or iris. 26 had reduced vision in one or the other eye, and 35 had reduced vision in both eyes. One patient was blind in both eyes as the result of sclerosis of the cornea (Fig. 3), three patients were blind in the left eye only; in two cases this blindness was caused by leprous involvement, in the remaining case the cause is doubtful. The accessory lacrimal gland was hypertrophied in 10 cases, 27 had leucoma of the cornea, 4 had corneal ulcers, and 17 had some other form of keratitis. The pupil was rigid and exudative iritis was present in 18

cases. The remaining cases of reduced vision were probably due to refractive errors, or should be explained by illiteracy combined with embarrassment at the time of the vision test.

The conjunctiva was reddened in 45 cases, and 34 patients gave a history of having had pain and redness in both eyes for a long time prior to my examination. 60 patients had facial tremor or fibrillation, and 27 had lid palsy (Fig. 4) or orbicular paralysis. The iris was immobile in 12 cases, probably due to paralysis of the sympathetic, since there was no evidence of inflammation, old or new.

10 cases had nodules on the lids, 5 had nodules on the globe at the outer limbus and 4 had nodules on the iris. 9 eyes had marked pannus formation and 4 had ptergyia. Of these cases 4 have had bilateral lid repairs.

The eyebrows were totally absent in 5, and the lashes in 6 mixed advanced cases. The brows were partially gone in 38 cases, of which 27 were mixed, 6 neural and 5 nodular.

My figures will doubtless raise the question as to whether the lesions of the iris and cornea might not be due to other causes than leprosy. I recognize that possibility. Syphilis has often been suggested as a factor in some of these eye conditions, and it is significant that our records show a frequency of miscarriages. The histories obtained from 80 women at Kalaupapa showed that 65 had children, and 22 had miscarriages; and of 25 married women at Kalihi, 13 had children, and 11 had miscarriages. It is my intention to carefully check over these patients with histories of miscarriages in order to obtain further statistical data, and with the idea of proving them positive or negative. While the task would be tremendous, and at the present time impossible, here it would be very valuable to have a Wassermann test of every leprous patient; particularly as they often give a history of more than a years treatment for syphilis, before their condition was diagnosed as leprosy. The best test of all is the therapeutic test, though iodides are apt to increase the cutaneous symptoms in leprosy.

While I have not touched upon the possibility of tubercular complications, that, too, is borne in mind. The only thing was to determine such points by more study and by more treatment. Working conditions at Kalihi are slowly being

improved, and when a place has been properly equipped for the purpose, it is hoped that I can study each case with more care.

My observations, up to the present time, at any rate, lead to the conclusion that the loss of vision in leprous patients is chiefly due to corneal changes and exudative iritis. The corneal conditions offer the greatest hope, since early treatment can do much. In cases of orbicular paralysis I try to instill into the patient the principles of hygiene, an understanding of which is the greatest single factor in conserving the sight in these exposed corneæ. I have done also, 101 lid operations, all after the technic of Fuchs, with good general success.

For the corneal margin blood vessel infiltration and marginal keratitis resulting therefrom, I usually do a peridectomy, resecting from 2 to 3*mm* of tissue, down to the sclera entirely around the cornea. With a sharp curette I then destroy the deep blood vessels not removed by the peridectomy. The result is very gratifying. In case of recurrence I repeat the operation, as I have done three times on several eyes with no apparent ill effects.

The ciliary affections—the exudative iritis, present the greatest problems. Here there is very little that can be done, especially if not seen early. In the early stages I instill atropine, and, if the sympathetic is not entirely gone, usually get a large pupil. In cases of long standing, where the pupil is pin point, filled with exudate, and bound down with posterior synechiæ, we often get brilliant results by performing an optical iridectomy. I have performed this operation about 27 times. 4 cases operated upon two years ago are still enjoying fair vision. Several others have had temporary improvement in vision, but the good effects from this operation unfortunately do not last long in the majority of cases. The inflammatory process begins anew, and there are more exudative deposits in the new pupil. Ofttimes the operation lights up the process immediately and the patient enjoys improved vision but a short time. In these cases we have complete failure.

I have had no success with medication for any other condition than the iritis, for which atropine and dionin, if used

early, accomplish much. I have tried subcutaneous injections of milk on 11 cases suffering from acute iritis and succeeded in lessening the pain in several.

In my early operative work upon the globe I made repeated attempts, using the technic commonly employed by most oculists for similar operation, to anæsthetize with cocaine solution 4%, instilled in the cul-de-sac. I found that cocaine was generally only partially successful, and often entirely useless. I now infiltrate every case with novocaine solution ½% except in the skin where I use 2%. In performing iridectomies I infiltrate circumcorneally allowing ample time for absorption to take place before making my section, which rarely causes pain. The patient frequently has pain when the iris is grasped, but I am usually able to perform the iridectomy without further anæsthesia.

I have no explanation to offer, except in the orbicular cases, regarding the failure of cocaine as an anæsthetic. Where orbicular paralysis is present, it might be that the failure is due to the ectropion allowing the solution to escape, and to the great dilution that takes place as a result of the epiphora.

I have observed that facial fibrillation always appears before ectropion or lid palsy takes place. I feel confident that facial fibrillation is the first step in orbicular paralysis, as I have noted (in cases which showed complete paralysis in subsequent examinations) that its presence is associated with a distinct loss of tone, especially of the lower lid. After examining several hundred patients, the examiner becomes adept in detecting early loss of muscular tone, much as one becomes adept in estimating the tension of the eyeball by the finger method.

These cases of lid paralysis are very distressing for, unlike iritis involvements, they are painless, and ofttimes do not drive the patient to you for relief until severe damage has been done. Most of these patients come from the lower walks of life and are accustomed to bearing mild inconvenience silently; symptoms of epiphora, purulent discharge, etc., do not disturb them, and often they appear for treatment only because of loss of vision.

Madarosis almost always occurs in the advanced or moderately advanced cases of all types, but its presence in the

mixed advanced type greatly exceeds its occurrence in all the others combined. This process begins in a small patch of indurated skin, almost always in the external or temporal region of the brow. This induration cannot be called a nodule, though it is often considerably thickened. The skin of the involved area becomes dry and anæsthetic, a dustlike scale appears, and shortly afterward a small patch of madarosis. Then another area appears, and thus, finally, the entire brow is destroyed. If a few hairs remain, as is often the case, they are usually at the nasal margin, but some cases retain a few scattered hairs. The lashes thin out gradually and do not disappear in bunches as the brows do. The lower lashes are usually affected first.

Several writers have described so-called nodules of the iris, but I doubt whether their descriptions are of real nodules. It seems to me that nodules anywhere on or in the globe must by their very nature be inflammatory. There is always much exudate and swelling, but I have never called anything a nodule of the iris, until I have ruled out the possibility that the nodule comes from the iris angle. It is here that we find the scleral nodule infiltration into the cornea, and it is this condition which ends in lepromatous granuloma of the cornea (Fig. 5). If I can look through the cornea and the exudate, and see the iris pushing forward or swollen, and if there is circumcorneal injection but no old or new nodules at the external limbus, I call it a nodule of the iris even though I cannot positively see the nodule itself. The little round masses the size of a pinhead, frequently seen in the vicinity of the circulus iridis minor, and so often described as nodules of the iris, are, in my opinion, exudates resulting from some previous iritis. In these cases the anterior chamber is not hazy, but there are often synechiæ of the iris. The masses do not grow but remain quiescent unless there be a new attack of iritis, as is very possible. These patients usually give an iritic history.

Some writers are of the opinion that nodules of the globe are secondary to nodules of the skin of the face and lids, and that they get on the globe by tissue continuity. While I have no figures to disprove this theory, I feel very strongly that it is not the case. If it were, why should we always see

the nodules of the globe at the external limbus, as far away as possible from the cutaneous structures? I have never seen what I considered a leprous process of the conjunctiva of the lids, the cul-de-sac, or the bulb,—except at the limbus (Fig. 6). Here we see the real nodules. I found the B. lepra teeming in 31 that I examined after dissection. These nodules are usually situated in the temporal limbus, slightly above the horizontal, and are usually accompanied by a ribbon-like induration or circumcorneal infiltration above. The nodules extend into and seem to be a part of the sclera. Dissection often arrests the process; in fact I have records of 8 cases operated upon two years ago, without recurrence. Dissection is often difficult because of the danger of perforating the globe. However, in the more advanced cases there is usually a line of cleavage or demarcation from the tissues beneath, extending forward into the cornea. When allowed to remain, these nodules infiltrate farther and farther into the deeper tissues of the cornea, until we have a large lepromatous process, or granuloma, which extends forward as an anterior staphyloma (Fig. 5). The mass protrudes forward between the lids. There is foul discharge, but little pain, and no sensation in the later stages; with the result that the mass may reek with filth, and be covered with flies and insects of which the patient is wholly unaware.

In the corneal conditions we often see multiple punctate spots, or areas, usually along the margins, but sometimes scattered throughout the cornea. While usually superficial, these may be deep. I have taken numerous scrapings from the cornea where these leucomatous spots are most frequent and in a few cases I have found the B. lepra.

I have examined the fundus in many cases, but I have observed no conditions that I thought might be due to leprosy. In all cases of reduced vision or blindness I have found ample causes in the anterior half of the eye. I have not seen a single case of optic atrophy which seemed due to leprosy, but in the two cases I found, the history of the patients suggested syphilis as a cause. Two other cases had old choroiditis; one patient stated that the condition had been present for many years, the other could give no history and escaped careful study.

CONCLUSIONS.

1. Every patient suffering from leprosy will probably have, sooner or later, some involvement of the eye.

2. The eye is more frequently attacked in the moderately advanced or advanced case, but the primary corneal changes may occur at any stage.

3. Exudative iritis is probably the greatest single cause of blindness.

4. Corneal changes, secondary to invasion from nodules in the ciliary region and to exposure keratitis, are next in causing blindness.

5. Facial fibrillation probably precedes most cases of orbicular paralysis.

6. Dissection of nodules from the limbus should always be done, since it at least gives hope of temporary arrest of the process.

7. Internal and external tarsorrhaphy is a distinct aid in conserving the cornea (Fig. 6).

8. Atropine in the early iritis is imperative.

9. Cocaine instilled into the conjunctival sac is unsatisfactory as an anæsthetic in leprosy.

10. Leprous patients seem to be immune to the ordinary pus producing organisms.

CHANGES IN REFRACTION; FROM A STUDY OF CASE RECORDS DURING FOUR DECADES.

By Dr. C. F. CLARK, Columbus, O.

(With four figures in the text.)

IN considering the variations which we encounter if we carefully measure the refraction of the eye at intervals during a long series of years, and in our endeavor to arrive at a fair understanding of the causes which account for these changes, we should bear in mind that, while apparently quite stable in its structure, the human eye is normally undergoing certain, slight changes during the whole period from birth to extreme old age and, while the line is not always easily drawn, we should endeavor to make, so far as that is practicable, a distinction between changes which are due to normal, physiological processes and those of pathological origin. Under normal conditions the eye at birth has an antero-posterior diameter of only 15.8 to 17.5*mm* while in the adult it is 24.3*mm*, a difference of 6.8 to 8.5*mm*. This difference in length of axis is largely made up by the growth of the eyeball during the first nine years of life by which time it has usually attained its normal size.

As is so well stated by Fuchs, "the infantile eye is one in which two opposing refractive states are combined, namely, a very considerable curvature myopia seated in the lens and a still more considerable axial hyperopia due to the excessive shortness of the eyeball." At the other extreme of life, in old age, we have a more rigid sclera, an inelastic, less spherical lens which cannot change its convexity with the action of the contracting ciliary muscle and we also have media which, while yielding under good illumination $\frac{5}{5}$ or even $\frac{5}{4}$ vision, have utterly lost what the photographers would call the rapid lens effect of the pellucid, perfect transparency of infancy.

It requires a very delicate adjustment of the curvature and the refractive index of the infant's lens to the gradually increasing length of the antero-posterior axis during the child's growth to avoid hyperopia on the one hand, or myopia on the other, when the eye has attained the normal, adult size at the age of ten, and when we consider the changes which must take place by the addition of new cells to the periphery of the lens throughout the life of the individual, it is almost miraculous that our refraction should be as stable as we find it. It is true, however, that, when we use accurate methods of measuring refractive errors, take the pains which such work requires and extend our observations over a long period of time we do find a great variety of changes and in explanation of these changes a variety of more or less plausible theories have been advanced.

Realizing, as we do, that in many cases the correction of even very low degrees of refractive error yield such admirable results in relieving asthenopia and various reflex nervous symptoms, the importance of the subject is established beyond dispute.

During the short period since I undertook the collection of material for this paper I have, in delving into the records of forty years of continuous practice, found so many cases of absorbing interest that I have realized that a proper study of these records would be so time-consuming that it would be necessary for me to be content for the present with what may be termed a preliminary survey and present only a few of the more typical and interesting cases which may be of some value in our efforts to arrive at a full understanding of this intricate subject. Some fifty or sixty years ago, when the refinements of the science and art of refraction, as we now know it, were being developed by the great pioneers in this important department of ophthalmology, though some variations in the degree and axis of astigmatism were deemed possible, such variations were looked upon as quite unusual. Hypermetropia with its variations was much better understood than astigmatism and the attention of the advanced workers was being directed very vigorously to the subject of myopia and the causes leading to its progress.

In Germany myopia of high degree was quite common and

there seemed to be a decided tendency to its increase in this country as revealed by the systematic study of refraction in the public schools and colleges. The distressing and often tragic results in the comparatively limited number of cases of what we would now term malignant myopia, in which the sclera shows a tendency to become thin and progressively stretch to such a degree as to amount to a posterior staphyloma, as it was pictured by the laborious studies of German and other continental pathologists, had made a deep impression on the minds of ophthalmic surgeons in this country and it is not strange that they should, for a time, have had an exaggerated idea of the relative frequency and the significance of these cases of progressive elongation of the eyeball and that some of them should have looked upon every instance of progressive myopia in the young as a potential case of what we are now inclined to classify as malignant myopia, and to treat it on that theory. I will have occasion to refer to this very important subject more in detail in connection with some of the cases I will present for your consideration.

How to present a group of long-drawn-out case histories and the figures in a long succession of refraction records without making them utterly boresome has to me presented a problem requiring some serious thought and I am by no means sure that I have succeeded in solving it.

When in the routine of the consulting room we find it necessary to look back over six or eight pages of closely written notes, or when we have a carefully tabulated group of refraction formulæ worked out for different periods of the life of a patient, it is often very difficult for us to carry in our minds an accurate picture of the sequence of events and to realize the exact significance of certain changes and their relation to other phases of the case in hand. For this reason I have been experimenting in what to me is a new field; the application of the curve or graph system to the study of the life history of my refraction cases. There may be some better system for accomplishing this same purpose and, if so, I would like to try it, but for two purposes I have found the plan I am employing admirable. First, it enables me, by entering on the chart the date of an important event in the patient's clinical history, to see at a glance its chronological relation to changes

in the refraction curve and, what I have found of great interest, it enables me at a glance to compare what might be called the refraction life history of one patient with those of others.

In the charts I have used the space between two of the fine, vertical cross section lines stand for one year of the patient's life while two of the heavier (or eight of the lighter), horizontal lines stand for one diopter of refraction error. I have only undertaken to work out this plan very recently and have as yet only a relatively small number of cases whose graphs are complete but they have brought out, to be seen at a glance, some very interesting points for comparison which by the old method would have required prolonged and concentrated effort. I am now having the refraction records in a number of my office case histories which, in the course of the decades have grown to be somewhat voluminous, tabulated and in some instances have had graphs made for my office use which are proving invaluable as time savers and as a means of obtaining a comprehensive record of the refraction changes and their relation to the other salient points in the patient's clinical history. With a little instruction, an assistant or stenographer can do practically all of this work and make it a pleasure to refer to an old record, which under the pressure of a busy day's work, would otherwise have been very burdensome. A reference to the graphs, perhaps, makes this method more clear.

In our cataract work we are all familiar with post-operative astigmatism and its disappearance, or reduction in degree after cicatrization is complete. In groove-like marginal ulcers which are extensive, and deep enough to involve Bowman's membrane, a similar effect is produced by a slight degree of flattening of the corneal surface, as is well illustrated in one of my cases, No. 11, though this undoubtedly is often very slight and may even escape detection as the cause of the astigmatism which follows. In this case which was under my observation and treatment for twelve years there were recurring attacks of severe keratitis and episcleritis apparently of tubercular origin which would yield only to the most active treatment including tuberculin which seemed throughout the whole period to be a most important factor in bringing about improvement.

The ulcers, while sometimes within the transparent cornea, were generally in the marginal zone and, on two occasions, there was quite extensive episcleritis strongly suggesting involvement of the ciliary body.

It would appear that the changes in the refraction came on with the cicatricial contraction following the healing of the ulcers and the clearing up of the exudates accompanying the episcleritis. The recurring marginal ulcers and episcleral exudates during the twelve-year period involved a great variety of meridians of the cornea and it will be noted that the axis of the astigmatism has also changed a number of times. It is of great interest to note that, with proper correction of the frequently changing refraction errors and adjusting the cylinders to the varying axes, it has been possible to give the patient almost perfect vision.

With the accompanying graph of the degree of the refraction errors I have included a figure indicating the varying axes of the astigmatism.

The most marked instance of this character which I have seen is illustrated by Case No. 13, reported in the *Transactions of the American Ophthalmological Society* in 1903. A locomotive engineer 42 years of age with a deep, groove-like depression paralleling the superior corneal margin throughout approximately 170° of its circumference and 3*mm* within the limbus, had an astigmatism of 22 diopters with uncorrected vision only sufficient to enable him to count figures at 4 meters. After I had curetted and thoroughly applied the galvano-cautery and cicatrization had taken place this was reduced to 6 diopters and his vision, with this correction, came up to $\frac{6}{6}-$, and was the same, with the same correction, when examined again 18 years later.

Such cases are extremely rare, but I am firmly of the opinion that cases like No. 11 are far more common than has been suspected. We correct the refraction errors of such cases but sometimes fail to study them attentively enough to recognize the cause of the changes. I thus failed to fully recognize their significance in this case until I had assembled and tabulated the various formulæ in preparation for this paper. Such a method of summarizing the data and having it in graphic form greatly simplifies the problem.

The refraction of the eye in direct, central, or what may be termed "foveal" vision is, of course, the refraction of the area limited by the margins of the pupil; and variations in this refraction are determined by:

A. Variations in the length of the visual axis of the eyeball resulting in hyperopia, myopia or emmetropia.

B. Variations in the corneal curvature.

C. Variations in the lenticular curvature.

D. Variations in the refractive index of the media:—cornea, aqueous, lens and vitreous.

Under the heading B (Variations in the corneal curvature) we have 1. Changes due to pressure, which is often transient in character and due to a variety of causes. 2. Relaxation in some portion of the cornea. 3. Cicatricial contraction in some portion of the cornea. 4. "Bow-like" curvature of the cornea in one of its meridians with a band of secondary membrane acting as a bow-string. Variations in the length of the eyeball producing varying degrees of hyperopia and myopia, with the grosser forms of variation in the corneal curvature, such as have resulted from cataract extraction, corneal cauterizations and other cicatricial changes in Bowman's membrane, have been studied with care and quite well understood for many years, a fair number of good illustrations of such changes being shown among the cases presented for your consideration, and on these cases, which are quite well understood, we need not spend much of our time, but there are many cases of variations in curvature refraction, sometimes of the cornea, sometimes of the lens and sometimes, it would seem, of both cornea and lens and, perhaps, complicated by changes of the refractive index, that are far more intricate and will well merit our careful study. Many cases of asthenopia accompany the earlier stages of presbyopia, and it is my belief that some of our cases of what we term incipient cataract, which are often accompanied by asthenopia, are associated with varying degrees and varying axes of astigmatism due to slight defects in the integrity of the lens which are only manifest when the lens becomes more dense and less responsive to the action of the ciliary muscle. Case 18 of anterior lenti-conus and Case 17 of injury of the anterior capsule by gunpowder seem to belong in this group

with Cases 15 and 16 of coloboma lentis which I have described in detail in papers presented to the American Ophthalmological Society and which may be found in the *Transactions* of that society in bound Vol. 7 for 1894–1896 and Vol. 17 for 1916.

It is not possible to go fully into this interesting subject, but I wish to call attention to the fact that in many of the graphs marked irregularity will be found in the refraction curve for myopia, hyperopia and astigmatism or a pronounced change in the axis of the astigmatism corresponding in time with the advent of presbyopia or the appearance of incipient cataract often of so low a degree or so far in the periphery as to have little or no effect on the visual acuity after the refraction error is corrected. This is well illustrated in Case 2 of advancing myopia and in Case 3, both of which have been under observation for more than thirty years and in both of which vision is still normal, the cataractous process, discovered many years ago, seeming to have been kept under control.

Only the limitations of our powers of observation and the limitations of accuracy of our measurements prevent us from solving the problems of lenticular astigmatism but, dealing with so accurate and delicate an optical instrument as the human eye, we may rest assured that for every error in refraction, however slight, there is certainly a physical cause which it is up to us to discover and correct if that is possible.

Changes in refractive error within a relatively short period of time, and especially changes in the axis of astigmatism, should lead us to critically study the patient's eyes and also his general, physical condition and mode of using the eyes to discover the cause of such changes.

These changes have a real clinical significance if our powers of observation are only acute enough to discover it, and systematic records carefully reviewed from time to time should assist us greatly in our effort to discover what this significance may be.

In looking over our records of patients past the mid-period of life we will often find the note: "slight opalescence of the crystalline lens, marked radiating strictions, some cortical opacities" or simply "incipient cataract." Some of these

cases go on to the completion of fully developed, senile cataract, and others, owing to a variety of causes which we sometimes are unable to determine, do not do so.

If all sources of focal infection are removed, prompt and proper elimination is established and rigorously maintained, proper habits of using the eyes are adopted, the varying errors of refraction are properly corrected as they arise and, in many cases, if what is sometimes termed mild alterative treatment is maintained at intervals for a long period of time, both the physician and the patient are often surprised to find that little, if any, progress is made in the development of cataract and, after ten or even twenty years have elapsed, such patients' vision will often remain without further material impairment. It is in such cases as these that a tabulation of our records of variations in the errors of refraction may be of great service to us and, if these records can be presented in graphic form, the relation between incidents in the patient's physical condition and mode of life and the state of the eyes can be shown in its true significance.

List and Summary of Cases.

1. O. R. Under observation 38 years. Myopia and myopic astigmatism, gradually changing in mid-life to emmetropia, and later hyperopia and hyperopic astigmatism. If changes in the lens are developing they must be in the direction of lowered, rather than increased refractive power.

2. H. Under observation 39 years. Myopia in a persistent student. Increase in myopia in spite of correction of a moderate degree of astigmatism. Increase of myopia from 19 years of age to 38.

3. B. Under observation 35 years. Increasing myopia notwithstanding frequent correction of astigmatism. The $\frac{5}{8}$ vision continues in spite of an incipient cortical cataract which was discovered at age of 42—twenty-six years ago.

4. X. Under observation 52 years. Period of record 36 years. Myopia and persistent asthenopia at 19. Uncorrected astigmatism. Increasing myopia for four years

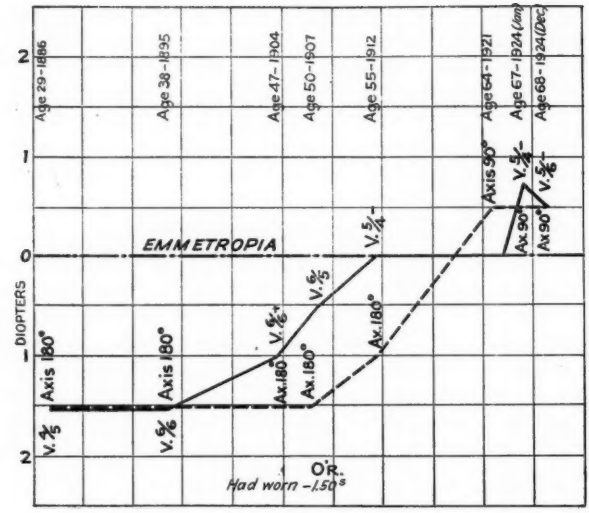

CHART 1.

when astigmatism was corrected, after which myopia ceased increasing, and, as in Case 1, advancing years brought diminishing myopia.

5. W. K. R. Period of observation 33 years. A moderate degree of hyperopic astigmatism ¼ D. to 1 D. No other refraction error and less than ¼ D. of variation in the degree of astigmatism in 25 years.

6. E. F. D. Period of observation 35 years. A moderate degree of hyperopia changing gradually to emmetropia and hyperopic astigmatism gradually increasing with the same axis 90° for 35 years.

7. D. F. H. Period of observation 35 years. Hyperopia remaining at .25 D. for 26 years and hyperopic astigmatism remaining at .50 D. axis 90° for 32 years. Illustrating the normal, stable condition of refraction seen in many cases of low degrees of hyperopia. Note that vision remains at or above normal at each examination.

8. G. H. Period of observation 38 years. Hyperopia of 2.25 D. in youth diminishing to 1.50 D. and hyperopic astig-

matism of .75 D. in the same period increasing to 1.75 D. and both remaining at these respective points for 24 years. The same axis of astigmatism (90°) persisted throughout

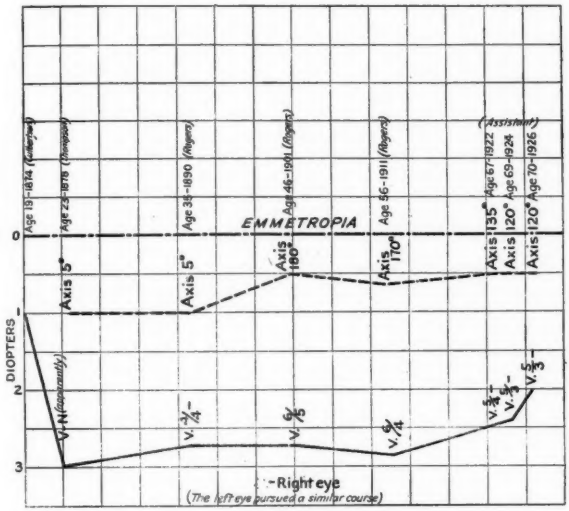

CHART 2.

the entire period of 38 years, as did normal vision. Note that between 43 and 49 years slight changes in the degree of both astigmatism and hyperopia are developing, the latter increasing and the former diminishing, the reverse of what took place in youth.

9. A. R. Period of observation 39 years. A moderate degree of hyperopic astigmatism remaining practically the same for 39 years. A moderate degree of hyperopia varying slightly, till the age of 42 after which it gradually increases.

10. E. L. D. Period of observation 33 years. A moderate degree of hyperopic astigmatism varying slightly from 45 to 50 years of age and then remaining the same for 26 years while the hyperopia increases 2 diopters. Marked asthenopic symptoms relieved first by general treatment, later by correction of refraction error, later still by correc-

tion of muscle imbalance only to recur and be again relieved by removal of an infected tooth.

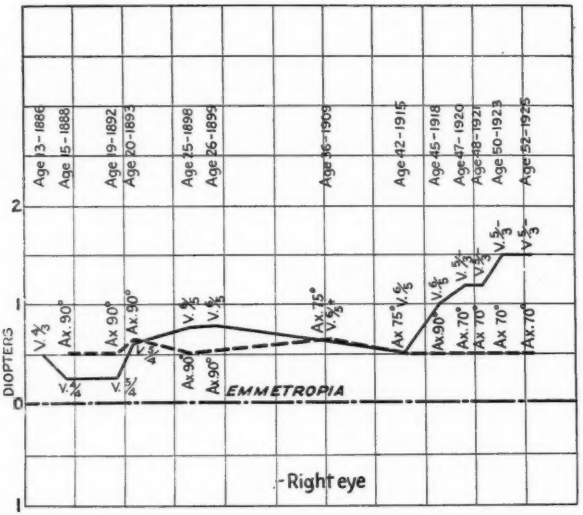

CHART 3.

11. C. P. B. Under observation 12 years. Hyperopic astigmatism increasing from 0.25 D. and later falling to 3.25 D. with marked variations in axis, due to recurring marginal ulcers apparently of tubercular origin. Hyperopia varying but vision when corrected remaining fairly good.

12. J. A. B. Under observation 3 months. Myopic astigmatism and myopia of 1 D. both accompanying a deep, groove-like ulcer near the inferior limbus of the cornea, the astigmatism disappearing at the end of two months after cicatrization of corneal ulcer. The myopia disappearing at the end of one month with the development (or reappearance) of hyperopia of 1.25 D. at the end of the 2 months.

13. E. M. R. Reported in *Transactions Am. Oph. Society*, Vol. 10, 1903–1904. A deep groove-like depression parallel with border of the left cornea causing 22 D. of astigmatism was curetted and the galvano-cautery used to

produce a cicatrix which reduced the astigmatism to 6 D., the correction of which brought the vision up from ability to count fingers at 4 meters to $\frac{6}{6}$ — vision which was found to be the same after 18 years.

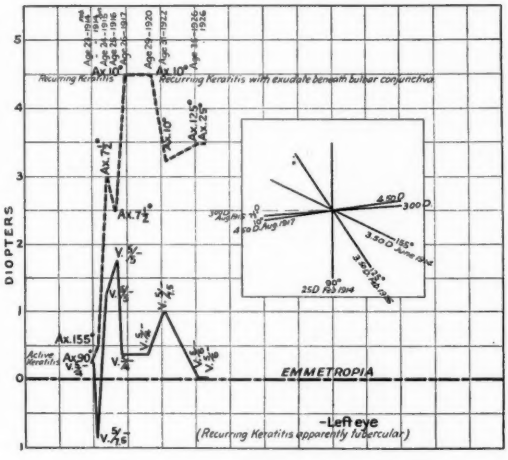

CHART 4.

15. E. S. Under observation 24 years. Coloboma o the lens of left eye, inferior portion. Myopic astigmatism of left eye 1 D. at age of 13 and increasing gradually until 5.50 D. at age of 34.

Myopia 2 D. at age of 10 increasing to 5.5 D. at age of 34. Axis at 90° at age of 13 to 15. After which it was consistently at 180°. Amblyopia (V. $\frac{4}{18}$) at 10. V. $\frac{6}{7.5}$ at 34.

16. M. H. Under observation 22 years. Coloboma of lens of left eye, inferior portion. Myopic astigmatism of 1.00 D. at age of 9, increasing to 4.00 D. at age of 31. Myopia of 0.50 D. at age of 9 increasing to 2.50 D. at age of 31.

Axis of astigmatism at 150° at 9 years and gradually changing to 175° at 31. Amblyopia (V. $\frac{6}{20}$) at 9. V. $\frac{6}{7.5}$ at 31.

It is to be noted that in both of these cases of coloboma there was rather marked amblyopia at the age of 10 and that later in life vision came up to $\frac{6}{7.5}$.

Is it not probable that the evolution of a lens with coloboma is a slower process than in the normal lens?

17. O. S. K. Under observation 29 years. Gunpowder deposited in the anterior capsule of the lens of the right eye. The eyes injured by the explosion of fireworks at age of 10.

Hyperopic astigmatism 0.50 D. at age of 11, increasing irregularly to 1.50 D. at age of 39. Hyperopia changing at frequent intervals and varying between .50 and 2.00 D. Some asthenopia at times but corrected vision normal or slightly above normal throughout the 29 years, excepting when a violent traumatism induced a choroidal hemorrhage in the macular region. The axis of astigmatism varied from 82½° to 105°.

18. G. W. Under observation 11 years. Anterior lenticonus. A well defined more or less symmetrical elevation near center of anterior surface of crystalline lens. Hyperopic astigmatism 1.50 D. to 4 D. Hyperopia varying from .25 D. to .50 D. later to emmetropia, finally varying degrees of myopia increasing to 1 D.

Considerable asthenopia at times but vision varied from $\frac{6}{6}$ to $\frac{5}{10}$ and $\frac{6}{6}$ back to $\frac{6}{6}$ and the patient was able to do her work as a high school teacher.

The axis of astigmatism varied 180° to 165°. There was some lenticular opalescence discovered in 1922 at about the time when the myopia began to develop and her general health led her, for a time, to give up her school work.

19. E. O'C. Under observation 8 years. Traumatic iridodialysis in the right, temporal quadrant by B. B. shot from an air gun. Re-attachment of iris within a few days. 0.50 D. of myopic, changing gradually to 0.50 D. of hyperopic astigmatism with hyperopia increasing to 1.00 D. in the course of the eight years. In spite of the rather severe traumatism in the region of the ciliary body and the irido-dialysis I could not assure myself of any serious injury to the lens, and vision remained at $\frac{6}{4}$ and $\frac{6}{6}$ from the time the blood cleared from the anterior chamber.

20. V. P. D. Under observation for 32 years. Incipient cataract apparently checked. Hyperopic astigmatism .75 D. with no hyperopia or myopia from 34 to 49. Myopia from 49 to 55. − .75 at 55 with advent of incipient cataract and some fine vitreous opacities. Myopia changing to .25 hyperopia at 56. No further change in astigmatism or hyperopia from 56 to 66. Corrected vision remains at

$\frac{5}{4}$– throughout 32 years. Axis of astigmatism changes from 90° to 100° and finally 120°.[1]

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[1] The cases briefly described in the summary are all illustrated by charts similar to those printed herewith, but it was not deemed necessary to present all the charts in this paper.

In the four charts the fine horizontal lines and vertical lines are one inch apart.

2 inches on the vertical lines = 1 Dioptre. 1 inch on the horizontal line = four years. Hyperopia and hyperopic astigmatism are shown above the ——— broad, horizontal, broken line which is marked "emmetropia."

Myopia and myopic astigmatism are shown below this "emmetropia" line.

Spherical errors (hyperopia and myopia) are shown by continuous heavy lines.

Astigmatic errors (hyperopic and myopic) are shown by — — — the broken line.

OBITUARY.

Alexander Duane, M.D., D.Sc.

Lieutenant, U.S.N., Honorary Surgeon Herman Knapp Memorial Eye Hospital, Chairman Section on Ophthalmology A.M.A., 1917, President American Ophthalmological Society, 1924, etc.

In the death of Alexander Duane, Ophthalmology has lost one of its most brilliant and enthusiastic leaders. He was by early training and natural inclination particularly well fitted for his work.

He was born at Malone, N. Y., on September 1, 1858. His father, General James C. Duane, was a graduate of Union College and West Point, and was later stationed at West Point in charge of engineering. His mother was Harriet Brewerton, daughter of General Henry Brewerton, superintendent of West Point, when his daughter was married to General Duane. His grandfather was James Duane, and his great-great-grandfather, Judge James C. Duane, of the Revolution, was the first mayor of New York City, and one of the founders of Union College.

At the time of the boy's birth, General Duane was in charge of the Utah Expedition, and neither he nor Mrs. Duane would ask for his exemption from duty. This same devotion to duty was a characteristic of Dr. Duane, in anything which he undertook, whether it was ophthalmology, writing, working for his college, or aid to some one he might befriend.

On the return of General Duane from Utah, he was stationed at West Point until 1861, when the family lived in Philadelphia, while General Duane was with the Union Army. At the close of the war they moved to Willets Point, N. Y.,

DR. ALEXANDER DUANE

UNIV. OF MICH.

and then to Portland, Maine. It was while a boy of eight to fifteen at these posts, that Dr. Duane developed his love for the water. He was a strong swimmer, a good sailor, and in later years nothing gave him more joy than a return to Portland to renew his boyhood habits. Because of the uncertain movements of an army officer, the boy was tutored at home, but his father always supervised his training in mathematics. His mother, who was equally well trained in English, chose for him, read to him, and guided his training in this subject. How well these were done is apparent in all the writings and research work of Dr. Duane.

He attended High School in Portland, and went from there to St. Mark's School. At the age of fifteen he passed his college entrance examinations and entered Union College, Schenectady, N. Y., in the fall. He graduated in 1878, at the age of nineteen, carrying off the major honors of his class, including the Phi Beta Kappa Key, which he always prized above all other honors.

His youth, scholarship, and spirit of good fellowship endeared him to his class. They were justly proud of him and he was beloved by all. After graduation he never missed a class reunion, unless prevented by illness. In 1914, his class presented him with a gold loving cup affectionately inscribed. This he held as one of his most choice possessions. The year following his graduation, was spent in the Albany Medical College, but the rest of his undergraduate training was at the College of Physicians and Surgeons, New York. On graduating from Medical College, he was interne at the New York Hospital, taking the full service. While there he began his literary work by writing the medical terms for Webster's Dictionary. He began practice in New York as assistant to Dr. Ranney, and later assisted Dr. George Stevens. He moved to Norfolk, Va., in 1888, and was there associated with Dr. Alfred Palmer. He soon returned to New York as assistant to Dr. Herman Knapp, with whom he worked for two and a half years.

His training in mathematics made him especially fitted for physiological optics, refraction and motor anomalies, and it is along these lines that he added most to the literature of ophthalmology. The precision learned through his math-

ematical training was, however, apparent in his adherence to detail in every branch of his work.

The first contribution to ophthalmology was entitled: "Some New Tests for Insufficiencies of the Ocular Muscles," and was published in the *New York Medical Journal* of August 3, 1889. In 1896 he wrote an essay on "A New Classification of Motor Anomalies," which was the prize essay of Columbia University. This classification was quite universally adopted, and although modified or changed in some minor respects, it is today the system generally accepted. In 1903, he translated the first American edition of Fuchs' *Text-book of Ophthalmology*, and this he continued until his death. Eight editions in all were published, the subsequent ones in 1908, '11, '13, '17, '19, '23, '24. On the completion of one edition, work was begun on the next. A system of note books was kept on the various subjects and in these were pasted or written, magazine articles, briefed articles, references to literature and original notes. From this accumulation of material, selections were made or statistics were drawn for the new edition. He derived much pleasure from his friendship with Dr. Fuchs, who, when in New York in 1922, made his home at Dr. Duane's. Their admiration for each other was mutual, as was also the enjoyment in each other's company. Thus a friendship, begun by correspondence, was sealed by personal contact. When the last, or eighth edition of "Fuchs" was begun, Dr. Duane had nearly completed the manuscript of a book on physiologic optics. This he never found time to complete. His work during the past year was chiefly on a book on Ocular Muscles, which was not finished. During this time, however, he contributed to the Jackson Memorial Volume, and to the various societies to which he belonged. His studies and writings on Accommodation have continued over a period of more than twenty-five years, as the first monograph was published in 1901. His last contribution on this subject was on the "Norms of Accommodation," in the Jackson Memorial Volume.

His contributions to Ophthalmic literature were: Translations of eight editions of Fuchs' *Text-book of Ophthalmology*. Medical terms in Webster's International Dictionary, Foster's Encyclopedic Dictionary of Medicine, Murray's New

Dictionary of the English Language, the Oxford Dictionary and the Students' Dictionary of Medical Terms, the latter published under his own name. He also wrote the chapter on Ocular Muscles in text-books by de Schweinitz and Randall, Posey and Spiller, and Weeks, and approximately seventy-five monographs on various ophthalmological subjects. Besides these, there were several non-medical subjects presented before the meetings of Phi Beta Kappa, and other societies to which he belonged or was invited as guest of honor. He also published two editions of *Rules for Signaling on Land and Sea*. This was used for some years as a text-book in the Naval Militia.

In return for his devotion and in recognition of his literary work, he was given the degree of Doctor of Science by his Alma Mater. In addition to the positions and societies mentioned, he was a member of the University Club, Hospital Graduates Club, American Ophthalmological Society, of which he served as president in 1924, The Ophthalmological Section of the American Medical Association, of which he was chairman in 1917, The New York Academy of Medicine and the County and State Societies, The Naval Reserve and the Spanish American War Veterans. He early joined the Naval Reserve and during the Spanish American War, was in charge of the signal service from Maine to Cape May. At the close of the war, he was discharged with the commission of Lieutenant, Junior Grade. During the World War, he served on board the training ship, *Granite State*. Here he trained details of men in signaling and quartermaster work. How well this was done is shown by the fact that practically all of the men under him were granted commissions. During this service he had the distinction of going and coming in the Navy as he chose, without an active commission and without pay. This service was continued daily from the time war was declared until its close.

He was keenly interested in amateur sports, especially those in which his Alma Mater participated. Any unsportsmanlike act, in any line of sport, met with his hearty disapproval, aptly expressed.

In 1891, he married Susan W. Galt, of Norfolk, Va., who with two sons, Robert Livingstone Duane, and William

Rogers Galt Duane, survive him. His eldest son, Alexander Galt Duane, was killed in service in France.

His home life was most delightful, and he was beloved by all whether related by blood or marriage. To those in need he gave of his time and money more than he could physically or financially afford.

The place of such a man cannot be filled. We value him and feel his loss, because of his breadth of mind and philosophical attitude toward his work. These, as well as his personal qualities, endeared him to all who knew him.

JAMES W. WHITE.

The outstanding regret connected with the death of Dr. Alexander Duane is that he was not personally more widely known to the medical profession, and especially to his colleagues in ophthalmology. The work he did for ophthalmic science has its established place in our literature, and will be appreciated by those who come after him. But behind that work was a man, whose character and personality influenced for good all who knew him. Contact with him taught modesty, patience and unselfishness, and elevated all one's ideals for the profession. His retiring disposition and unaggressive attitude toward all he met made it easy to undervalue the importance of what he had thought out and was ready to teach.

When a new idea occurred to Dr. Duane he did not proclaim its importance without giving it solid support, and so provoke opposition and unprofitable discussion. He first considered it very carefully, giving it much thought, reading what might throw light upon it from related topics, working it out experimentally, or determining what support was given to it by his own observations, or by those of others recorded in the literature. When he was convinced of its truth he published it, in a clear statement with the facts and observations that supported it, and without any claim for its importance or his own priority of discovery, appealing chiefly to the more thoughtful of his readers, who should in due time perceive and teach others its importance and correctness.

His name was not attached to operations or methods that would make it familiar to the mass of his colleagues; and often years passed before justice was done to his substantial contributions to our methods of practice. But, gradually what he did came to be appreciated; and high respect has developed for the man who discussed with clear intelligence and balanced judgment the motor anomalies of the eye, the changes of accommodation, the importance of good vision in the Navy and other topics. Appreciation had come to him in the late years, and was extending to all who came to know him personally, or who gave careful perusal to his writings.

A wide reader, compiler of dictionaries, author and translator, he never lost his interest and appreciation of the human side of student life. As alumni trustee of Union College, member and head of its student council, member of his fraternity chapter organization, he showed the same devotion to the general welfare of college students and their college sports, as he did when he played on the college baseball team. Duane had a broad view of medicine. He never lost that view in the treating of his particular subject. All that he wrote is worth reading. He has had, and will have, many readers. But those who knew him personally got from him something of high value, not to be transmitted by the printed page.

EDWARD JACKSON.

From a Very Old Friend.

Having enjoyed an uninterrupted friendship with Aleck Duane from his early boyhood, I am very grateful for this opportunity to record my appreciation of that friendship and of the rare beauty of his character. He was one of the most lovable fellows I ever knew and an ever delightful companion.

Along with his devotion to his chosen profession and his great achievements in it, he retained a living interest in Greek and Latin, which to me, as an old Latinist, was especially gratifying. Sometimes he would send me Latin versions he had made of little modern things. One particularly clever one was of that once popular ditty, "Everybody Works but

Father," which brought much entertainment to a number of us.

He took great pleasure, also, in good music, whether vocal or instrumental, and the various occasions on which we enjoyed such music together are among my pleasantest recollections of these later years.

HENRY PREBLE.

In the death of Alexander Duane the medical profession has suffered a grievous loss, and the science of ophthalmology is deprived of one of her most distinguished interpreters.

His scholarly attainments were preëminent; he respected his material; it left his hands finished with a master's touch.

His recorded studies of the anomalies of ocular motility constitute a notable and permanent chapter in this difficult and important department of ophthalmic science.

His clinical contributions to ophthalmology, always of merit, were characterized by a clarity of expression which greatly enhanced their value and revealed his highly intelligent grasp of the subjects they were designed to portray.

A gifted teacher, his students and colleagues became beneficiaries of the careful and satisfying work which was evident in all of his efforts.

His literary attainments were of high order; his translation and editorship of succeeding editions of the world's most famous Textbook of Ophthalmology placed English-speaking ophthalmic surgeons deeply in his debt.

The duties which pertained to his Trusteeship in Union College and to the welfare of the students were performed with fine fidelity.

As a citizen, he was true to the cause of civic righteousness and his service in the United States Navy was of conspicuous value.

Possessed of unusual talents, he was modest in deportment and gentle in his judgments. Correct in his relations to his colleagues and compatriots, he was a model of the ethical life of our profession. He will be missed as few men are.

GEORGE E. DE SCHWEINITZ.

My acquaintance with Dr. Duane began in 1885, when we were both engaged in the study of ophthalmology under the

guidance of the late Dr. Herman Knapp, in the Ophthalmic and Aural Institute, and our friendship continued uninterruptedly up to the time of his demise. He was an earnest, industrious and intensely interested student, always modest, always kindly, always true. An outstanding characteristic in Dr. Duane's character was his generous consideration of his colleagues. Although very busy with his own work, he lent a willing ear to the requests of colleagues for assistance. When, after a colleague had disappointed, I appealed to him for a chapter on the mascular anomalies of the eye, to appear in a treatise that I was endeavoring to produce, he said: "Weeks, my time is pretty fully occupied, but I will endeavor to accommodate you," which he did. Dr. Duane was true to himself and true to others. If consulted by a patient whose condition was such that he felt that the services of a colleague would be of more benefit to the patient than his own, he did not hesitate to request the assistance of that colleague. Not a few operations were turned over to other men because of such conviction. While tenacious of his position when satisfied that he was right, he was always willing to concede error when convinced that his position was not tenable.

A valued friend and colleague has passed beyond.

JOHN E. WEEKS.

REPORT OF THE SECTION ON OPHTHALMOLOGY, NEW YORK ACADEMY OF MEDICINE.

By Dr. THOMAS H. CURTIN, Secretary.

MEETING OF OCTOBER 18TH, 1926. DR. CLYDE E. McDANNALD, CHAIRMAN.

Dr. L. W. Crigler presented a case of **Unilateral exophthalmos, with choked disk.**

The patient, aged 25, with a negative history, about four months ago, noticed gradual protrusion of her right eye, accompanied by occasional diplopia, without pain or redness. The right eye appears to be pushed directly forward by an unyielding mass in the orbit. The motility of the eye is not disturbed. The exophthalmometer shows a proptosis of 8*mm*. The nerve head presents a swelling of six diopters, with little or no retinal œdema and no hemorrhages. The blind spot is slightly enlarged and there is a contraction of the upper and inner fields of vision. X-Ray and serological tests are negative. There are no evidences of constitutional disturbances. The tentative diagnosis is intradural myofibroma and it is proposed to remove the tumor at an early date by resecting the outer wall of the orbit (Krönlein).

Dr. Henry H. Tyson presented a patient who had an **Orbital neoplasm cured with radium and followed by cataract.** L. N., aged 26 years, was first examined December 1, 1923, with the history of a protruding left eye for two months and pain during the last two weeks. Left eye proptosed 13*mm* (exophthalmometer R:17, L:30) and 6*mm* lower than O. D. No bruit. Motility diminished in all directions. Pallor of disk. Some apparent thickening and resistance felt upon palpation under upper and outer part of orbit. General health good. X-ray of nasal sinuses negative. Vision

O. D. $\frac{20}{20}$, O. S. $\frac{20}{70}$; lenses clear. Tonsils and adenoids removed six months previous. Teeth negative, T. B. negative. Repeated Wassermann, blood and spinal fluid negative. As she had received anti-luetic treatment without benefit, a choice of a Krönlein operation or radio therapy was advised. She preferred the latter and was referred to the Memorial Hospital where, under the successful treatment of Dr. Quick, she began to improve gradually, the recession of her eye beginning after the third radium exposure during a period of five weeks. During the following five months, three more exposures were given and the proptoses entirely disappeared. Five months later she had a final exposure on October 21, 1924. Vision $\frac{20}{20}$; both fundi normal and motility normal, no exophthalmos. On September 26, 1926, she called for examination and stated that for the past four months her vision appeared to be getting poor gradually in her left eye. With a dilated pupil a sub-capsular opacity could be seen at the posterior pole and a white dot in the anterior part of lens cortex at ten o'clock. Also faint radiating lines from the opacity in cortext extending to periphery. Vision with correction R: $\frac{20}{20}$. L: $\frac{20}{20}$. Exophthalmometer R: 20, L: 20, no proptoses. Loss of hair over outer half of left eyebrow.

This case is of interest on account of an apparent cure (clinically regarded as a sarcoma) as a result of radio-therapy, and on account of the appearance of the lenticular changes occurring as sequelæ of the radium treatment, although all possible precautions were taken to prevent any deleterious effect on the ocular tissues. While others have noted similar changes, there have been few recorded cases; and some doubt exists as to the possibility of radium causing these changes. Dr. Tyson's opinion is that there is no doubt as to the radium being the primary cause of the changes noted.

Discussion: Dr. Schoenberg doubts if it is a sarcoma. As to cataract after radiation, he quoted a case of Dr. Verhoeff's of glioma where the X-ray was used from the side of orbit, the front of the eye being screened to prevent the formation of cataract. He also reports a case of opacities occurring in the posterior part of the lens in front of the capsule following the use of radium, the lens later becoming entirely cataractous.

Dr. QUICK states that he has seen a few cases of cataract formation following the use of X-ray and radium, but he feels that they bear no relation to the amount or manner in which the radium is used. He reports a case of Dr. Knapp's treated for lid tumor with protection of the eye and lens, and doubts if it is possible to protect the lens against major dosage, for the metal itself gives off secondary radiation and also scattered radiation. With our present knowledge, it is impracticable.

Dr. KNAPP: The case referred to by Dr. Quick has the same changes as Dr. Schoenberg's case. The lesion of the lid was at the outer angle and the opacity in the posterior cortex was on the outer side. He has had the opportunity of seeing a number of cataracts following radium; they were nuclear or posterior cortical and came on many months after radiation.

Dr. CRIGLER reported a case of cataract formation iritis and secondary glaucoma with final loss of the eye after two or more massive doses of radium.

Dr. LAMBERT stated that he had had five lid cases treated by Dr. Abbe; and in no case were there any changes in the lens.

Dr. SCHLIVEK spoke of four cases in which cataract developed following exposure to radium. In one, cataract developed over night with acute glaucoma. In cases where radiation can be avoided, it should be done.

Dr. DAVID WEBSTER reported a case of epithelioma of the lower lid which was treated by Dr. Abbe. No cataract developed. The case was one of six years' standing.

Dr. TYSON, in closing the discussion, stated that the patient had received seven exposures to radium emanations during the course of a year—totalling 10 hours and 22 minutes. No. of mc., 7,013.—Filter *½mm* silver + *2mm* brass.—Dist. *3cm.*—Location ant. to orbit (4), side orbit (3). Method of application brass tray. He also referred those interested in the effect of radium on the eye to a paper by Dr. Laura Lane in the Section on Ophthalmology, A.M.A., 1925, in which Dr. Curtin reported two cases of cataract induced by radium after exposure.

Dr. TYSON presented a case of **Removal of the lens in high myopia for improvement of vision.** S. B., aged 22 years,

came to the Knapp Memorial Hospital on March 2, 1920, with the request for an operation to relieve his nearsightedness. With $\frac{2}{60}$ D. S. = $\frac{2}{70}$. March 12, 1920, discission was performed on the left eye, the lens being found to be of a sticky nature. No reaction occurred; the crucial incision in the lens capsule closed. On April 8, 1920, the lens was incised deeply and four days later the posterior layers of cortext became cloudy. Eye white, tension normal, no cortex in anterior chamber. Lens continued to opacify slowly, and in April, 1925, a combined extraction was performed with uneventful recovery. Present vision O. S. −1.00−3.c O. = $\frac{20}{25}$. In the right eye only one needling has been performed and is still under treatment. Vision $\frac{20}{70}$ without glasses.

DISCUSSION: Dr. LAMBERT reports 20 cases of lens extraction in high myopia in which all but two results have been good (subsequent detachment of retina), one due to traumatism and one to a de Wecker operation.

Dr. WEBSTER has been completely converted to the Ziegler "Through and Through Incision."

Dr. HARTSHORNE has operated on two congenital cases of high myopia in which only one needling was necessary.

Dr. CURTIN has performed this operation on eight patients with excellent results. They consisted of both young and middle-aged patients, some with choroidal stretching and others not. All were able to return to useful work after being compelled to relinquish or modify their work on account of their eyes. His procedure is a series of needlings followed by linear extraction if necessary. He believed also that the myopia should be close to 20 diopters. Two cases of children, which were progressive before operation, appeared to be stationary following, being observed for five years.

Dr. CRIGLER has operated on a number of high myopias. He has succeeded in removing the lens after a series of needlings. He has never done linear extractions, although two patients were over thirty-five years of age.

Dr. MARTIN COHEN reported a case of **Lymphangioma of orbit,** illustrated by drawings, specimen and lantern slides. The history was given at the last meeting; but since then Dr. Cohen has performed a Krönlein operation and a full report is to appear.

DISCUSSION: Dr. KNAPP. If this tumor was within the optic nerve sheath, why were there no changes in the nerve head? And is it not remarkable that it could be so easily removed?

Dr. COHEN: Dr. Knapp's point is well taken: the mass, however, seemed to have a pedicle.

Dr. MARK J. SCHOENBERG presented **An apparently cured case of glioma of the retina**. (To be published in full.)

DISCUSSION: Dr. QUICK has had, on the whole, only unhappy experiences with the use of radium in glioma of the retina.

Dr. WALDSTEIN: Opacities of the vitreous in retinal glioma have been seen for a long time. Multiplicity of tumors is interesting and hard to explain.

Dr. JOSEPH S. HORY (by invitation) presented a spot in the fundus which was possibly a **melanoma.**

DISCUSSION: Dr. KNAPP thought this case probably one of retinal pigmentation.

Dr. MCDANNALD had seen a similar case which he diagnosed as nævus.

Dr. CLYDE E. MCDANNALD presented a case of **Retinal detachment with unusual features,** in a woman, aged forty. The detachment had existed for about six months, and the interesting feature was that it was covered with many grayish punctate spots superficial in character. Tension, transillumination, Wassermann, urinalysis and teeth were negative.

DISCUSSION: Dr. KNAPP was of the opinion that the spots were albuminous deposits.

Dr. MCDANNALD also presented a case of **Bilateral evulsion of the optic nerve.** A bullet entered the right temple and emerged from the left temple. A mass of connective tissue replaced the optic nerve heads.

The paper of the evening was **A classification of retinal diseases—retinitis externa and choroiditis,** by Dr. Manuel U. Troncoso. Dr. Troncoso believes that a new classification of certain affections of the retina and of the choroid is needed in order to clarify the confusion arising from the use of the terms chorio-retinitis and retinitis. Retinitis as applied indiscriminately to all affections of the retina is a misnomer; a more accurate term, except where there is a distinct evi-

dence of inflammation, would be retinosis (osis—condition of). Affections of the inner layers of the retina would then be classified as internal retinosis. Those which primarily involve the outer layers and are confined for a long time to that portion of the retina would be grouped under the heading of external retinosis. When both the choroid and retina are involved, there is chorio-retinitis. When the choroid alone is affected, especially in the early stages of the disease, the term choroiditis is properly applied.

The pathology of external retinosis is then described and its differential diagnosis with choroiditis. External retinosis is divided into the six following groups which are described in full: (1) Changes due to hereditary syphilis, (2) pigmentary retinosis (non-luetic), (3) pigmentary retinosis without pigment, (4) retinosis punctata albescens, (5) macular pigmentary retinosis, (6) hereditary hyaline infiltration of the retina (Doyne).

Chorio-retinitis Dr. Troncoso divided into the following types: (a) chorio-retinitis syphilitica (Foerster), (b) chorio-retinitis myopica, (c) chorio-retinitis sympathica, (d) chorio-retinitis by congenital lues (pseudo-albinism), (e) atrophia gyrata (Fuchs), (f) photogenous actinic. Finally, the subject of choroiditis is treated.

DISCUSSION: Dr. SCHOENBERG thought that the term retinosis was a good addition to ophthalmological words. It implies no inflammatory condition, and is analogous to nephrosis and many other words now used in medical nomenclature. Classification is simply a point of view. Dr. Troncoso's classification would appeal to pathologists particularly, but not to clinicians. He has tried to divide retinitis into various groups depending on their pathological situation—retinitis externa and interna, as has been suggested before. This, however, is not a clinical classification which appeals to the speaker, and he favors the old classification, until new developments arrive in the field of ophthalmology.

REPORT OF THE TRANSACTIONS OF THE OPHTHALMOLOGICAL SECTION OF THE ROYAL SOCIETY OF MEDICINE.

By Mr. H. DICKINSON, London.

The opening meeting of the session took place on Friday, October 8th, when Sir Arnold Lawson inducted his successor, Mr. Ernest Clarke, C.V.O., F.R.C.S., in a speech of cordial welcome.

Clinical Cases.

Mr. M. S. Mayou sent a case (which was shown for him by Mr. Cunningham) of massive detachment of the retina. At first there were hemorrhages on the surface, but now the sites showed pigmentation, and Mr. Mayou's view was that it was a massive exudate.

Mr. Mayou also showed a case of essential shrinking of the conjunctiva which might be due to pemphigus. The boy had been seen by three expert dermatologists, only one of whom thought the boy had had pemphigus. A picture of the eruption was shown.

The President asked for fuller particulars of the first of the cases, and remarked on the presence of a patch of old choroiditis.

Mr. Cunningham replied that Mr. Mayou's view was that the case was one of Coats' disease. No history of injury was obtainable. He, the speaker, had only seen the boy once. His condition was discovered at a school clinic in 1923. Vision in that eye at that date was $\frac{6}{18}$, and the sight had gradually deteriorated since. The deatchment was discovered only a month ago.

Dr. HUGH THOMPSON brought forward a case of electric retinitis, a rare condition. It was probably identical with eclipse blindness. A member suggested to him that the stippling in each macula might have been present already, that it might not have been due to the flash; but the exhibitor considered that the flash was the cause. A fellow workman at the same job also had blindness, but that was only temporary. In the three months since the accident this present patient had not improved. He had not discovered any central scotoma.

Mr. MONTAGUE HINE showed a case of neuro-fibromatosis of the right upper lid, in order to seek advice as to the wisest treatment. In March he attended hospital as his eye was hurting him, and there was a discharge from it, which he found it difficult to check. There were ulcerations of the lower lid, where the upper lid overlapped it. A section from the lid, taken in 1911, showed neuro-fibromatosis. The right eye was now practically blind, *i.e.*, mere perception of hand movements. The left eye was quite healthy. A similar condition was found in the patient's temporal region, and there was a small lump on the back of his head. He had thought of removing the eyeball and taking upper and lower lids away, giving the patient epitheliated sockets, as was commonly done in the War, but asked if a less drastic method could be recommended.

Mr. TREACHER COLLINS spoke of Mr. Simeon Snell's treatment of cases of the kind. In some of them Mr. Snell removed a large piece of the eyelid throughout its thickness, and there resulted a great improvement in the patient's appearance. One of Mr. Snell's cases had buphthalmos and the eye was removed and sent to the speaker for a pathological examination. He found that the ciliary nerves were involved as well as the nerves of the eyelid. In addition the choroid was remarkably thickened, and in the sections of the choroid were bodies which looked like enlarged nerve end-organs.

Mr. F. JULER referred to a similar case which he showed to the Section six years ago, and in which both lids were affected. In the worse eye of that patient the lid protruded markedly. At first the other eye was thought to be buphthalmic, but afterwards it was concluded that it was only

highly myopic, therefore the question of excision was left in abeyance. Large portions were cut out of both lids in a wedge shape. The condition extended to the temporal region, and much of the growth there was also removed, with definitely good cosmetic result.

Mr. HUMPHREY NEAME showed a case of interstitial keratitis with marked radial corneal deep striation. The condition occupied three-fourths of the area of the cornea. The striation was symmetrical, leaving the center more or less clear. It was probably of congenital syphilitic origin; Wassermann was positive, but there were no syphilitic stigmata.

Mr. TREACHER COLLINS thought the striations were due to rucks in Descemet's membrane, and the PRESIDENT said he had a case of the kind which was generally regarded as specific, but it turned out to be tubercular.

Mr. J. F. CUNNINGHAM exhibited a patient with a mass obscuring the disk. The boy came to hospital in 1920 as an ordinary case of refraction error. The left eye was defective, and the father said he was anxious about the child, as another boy had lost an eye from glioma of the retina, and an uncle had the same disease. He said the boy had been struck in the eye with the handle of a scythe nine months before he was seen by the exhibitor. The case seemed to come into the category of evulsion of the optic nerve.

Mr. DAVID WILSON (Torquay) showed a patient who, by violent coughing, had dislocated his lens into the anterior chamber. The doctor had been treating him for conjunctivitis. When, a fortnight later, the exhibitor saw him, he had glaucoma. As the eye was blind, it was removed, and the specimen showed a cataractous lens lying in the anterior chamber.

Mr. RAYNER BATTEN showed, on the screen, pictures of cases of Tay's choroiditis.

Address of the New President.

Mr. ERNEST CLARKE then delivered his address, which consisted of a review of the advances which have been made in ophthalmology during the fifty years immediately passed,

forty-five of which years Mr. Clarke had himself passed in its practice.

It was in 1881 that he attended the clinic of Donders at Utrecht, and saw Snellen demonstrate his scheme for detecting malingerers by causing them to read red and green letters through red and green glasses. The present universal employment of cocaine, novocaine, and allied substances rendered it difficult to realize that it was as recently as 1884 that Koller, of Vienna, first introduced cocaine to the profession. But the President uttered a caution concerning its possible misuse. Pain, he reminded the meeting, was nature's great indicator, and mere allaying of pain, without trying to discover its cause, was not good practice.

Perhaps the greatest change in the specialty in the period concerned was in the treatment of errors of refraction and of muscle-balance. In earlier days the material at the command of the refractionist was very limited, even though the use of the shadow test had been established. When he first attended Moorfields, there was only a limited range in the lens cases. In that period eye-strain was known as asthenopia, and only the single symptom headache was attributed to it. Mr. Clarke was inspired to write his book on eye-strain, in 1881, by reading the work of Gould, of Philadelphia. It was now realized that there was probably no functional nerve trouble of which eye-strain might not be at least a contributing if not the sole, cause. By attending to this it was possible, he contended, to indefinitely postpone epileptoid attacks, migraine, recurrent iritis, and perhaps to delay or postpone the appearance of glaucoma and cataract. Eye-strain was more likely to be associated with small errors of refraction than large; ⅛ of a diopter might make all the difference between efficiency and unfitness. The small wastage of effort in eye-strain was continued throughout many hours daily, and the effect of this was insidious.

Mr. Clarke did not agree with those who said that cycloplegics were not needed when estimating the refraction of an eye. He considered that most people of under forty years of age were unable to completely relax their accommodation, hence the observer could not get down to "bed-rock" in estimating the real condition of the eye. Certainly in all young

patients it was very important to paralyze the accommodation. And as the pupil was dilated during the examination, a post-cycloplegic examination was often required before writing the prescription for the glasses. Dr. Hawley, of Chicago, truly said that the main object of using a cycloplegic was to ascertain the exact degree of astigmatism.

In the treatment of myopia there had certainly been advance in the fifty years. In the earlier days people with no more than 3D. of myopia were allowed to do their near work without glasses; but it was found that the ideal treatment for myopia was full correction, at the same time correcting any astigmatism present; and, by forcing the ciliary muscle to do its proper work, stay the condition. He thought Sir Arthur Keith was correct when he said that myopia was due to a badly constructed sclerotic, and that this was prenatal and inherited.

Presbyopia formerly received very inadequate treatment, namely, by prescribing a plus glass, generally of the same strength for both eyes; the astigmatism was not corrected, unless it was extreme. Yet it was at the presbyopic age that eye-strain, if present, did most harm, as there was not then the same power of recuperation as when younger, and the general vital powers had begun to ebb. He strongly recommended bi-focal glasses for the condition, especially as they were now so skilfully made. There was a tendency, however, to make the reading portion too strong, so that anything beyond the strict focus was liable to be blurred. Correct centering of the glasses was of prime importance.

Maddox, of Bournemouth, had put the treatment of imbalance of the extra-ocular muscles on a sound basis, and his glass-rod test was an expeditious and easy method of diagnosing heterophoria. Small degrees of exophoria or hyperphoria might cause as much eye-strain as did small amounts of astigmatism; indeed the two were often associated. By careful correction of the refraction, the heterophoria could be gradually removed. Excepting in old people, or those who had a large error, prisms were not the best form of treatment.

He referred to the great value of X-rays in diagnosis and treatment; they gave a view of the bones of the skull, and revealed abnormalities associated with the pituitary gland,

besides enabling foreign bodies in the eye to be seen and accurately located. The value of radium, ultra-violet light and vaccine therapy was dealt with, and reference was made to the work of Goulden and others in successfully employing the ultra-violet light to the treatment of tuberculosis of the iris and ciliary body. Hepburn and Browning had shown how important it was, in sympathetic ophthalmitis, to do a differential blood count. Mr. William Lang had persistently called attention to the frequency of dental sepsis as a cause of eye trouble, others being tonsillitis and accessory sinus trouble. On the surgical side, Elliott's trephine operation remained the most popular for simple chronic glaucoma.

In the treatment of convergent strabismus in children great improvement had come about. By inducing parents to bring their children at an earlier age, many of these cases were now being cured by glasses alone, lessening thereby the proportion of operations for squint.

The surgery of the eye in war conditions was very efficiently met by Sir William Lister, helped by Mr. J. F. Cunningham, and the special precautions in isolation, etc., for which the former was responsible, resulted in the conservation of much of the Eastern fighting force, among whom trachoma was generally rampant.

Emphasizing the important part of medicine in the training of the ophthalmologist, Mr. Clarke recommended that those who had started serving as clinicals in an eye hospital should, if possible, spend at least a year in general practice. He proceeded to refer to improvements in the instruments now at the command of the ophthalmologist. He thought it would yet be years before the slit-lamp yielded all that it was capable of teaching.

In a few words concerning the future, the President hoped the causal organisms of trachoma and vernal catarrh would be discovered, and that a true theory of accommodation would be produced. Much more remained to be learned as to the significance of abnormally high or low blood pressure, and he spoke of the importance of registering both the systolic and the diastolic pressures. More knowledge was also needed as to the effects of endocrine glands in health and dis-

ease; Mr. J. H. Fisher and Dr. de Schweinitz had already given much help in connection with the pituitary gland.

In conclusion, Mr. Clarke said the mere realization of what ophthalmology meant provided a stimulus to further advance, and to that he looked forward with much hope.

On the proposition of Mr. Treacher Collins, seconded by Mr. Cyril Walker, Mr. Clarke was cordially thanked for his address.

REPORT ON THE PROGRESS OF OPHTHALMOLOGY.

Abstracts by Drs. A. N. Alling, New Haven; P. G. Doyne, London; and K. Wessely (*Archiv für Augenheilkunde*), Würzburg.

Edited by Matthias Lanckton Foster, M.D., F.A.C.S.,
New Rochelle, N. Y.

(*Continued from November issue.*)

IV.—THE ORBITS AND ACCESSORY SINUSES.

63. Cucco. **Dermoid cyst of the orbit.** *Annali di Ottalmologie e Clinica oculistica*, April-May, 1923.

64. Delord. **A case of sarcomatous metastasis in the meninges with bilateral exophthalmos and sinus thrombosis.** *Annales d'oculistique*, clx., 1, p. 16.

65. Elschnig. **Pseudotumor in the orbit caused by an encapsulated splinter of wood.** *Klin. Monatsbl. f. Augenheilkunde*, lxxii., p. 350.

66. Engelking. **Symmetrical tuberculomata of the orbit.** *Ibid.*, lxx., p. 100.

67. Filatow. **Removal of malignant tumors from the orbit.** *Archiv. f. Augenheilkunde*, xciii., p. 47.

68. Franklin, W. S. and Cordes, F. S. **A case of orbital lymphangioma.** *Journal of the American Medical Association*, lxxxiii., p. 1741.

69. Kubik. **The influence of exophthalmos upon the action of the obliques.** *Klin. Monatsbl. f. Augenheilkunde*, lxxi., p. 237.

70. Locke, Charles Edward. **Intracranial arteriovenous aneurysm, or pulsating exophthalmos.** *Annals of Surgery*, July 4, 1924.

71. Lodato. **Radiologic investigation of compression atrophy of the optic nerve.** *Bolletino d'oculistica*, January, 1923.

72. Maggiore. **Thrombophlebitis ophthalmo-cavernosa.** *Annali di Ottalmologia e Clinica oculistica*, April-May, 1923.

73. Di Marzio. **Cavernous [angioma [of the orbit.** *Ibid.*, January, 1923.

74. Morelli. **A case of menstrual exophthalmos.** *Bolletino d'oculistica*, August-September, 1922.

75. Ridley. **Notes and specimens of a case of intraorbital neoplasm.** *British Journal of Ophthalmology*, vii., December.

76. RING, G. ORAM. **Orbital cavernous hæmangioma with pulsating exophthalmos. Removal by electrocoagulation.** *American Journal of Ophthalmology*, December, 1924.

76A. STIEREN, EDWARD. **Melanosarcoma of Choroid with Extension to Orbit.** *Jour. of A. M. Assoc.*, Vol. 85, p. 1213.

77. WRIGHT, R. E. **Tumor of Orbit of Doubtful Origin.** *American Journal of Ophthalmology*, October, 1925, p. 781.

78. YERGER, C. F. **Exophthalmos a Complication of Nasal Sinusitis.** *American Journal of Ophthalmology*, November, 1925, p. 882.

CUCCO (63, **Dermoid cyst of the orbit**) reports three cases of this nature, in each of which the tumor was removed by Cirincione's operation. Cucco calls attention to the fact that the often very large cysts may extend into distant parts of the orbit, and that during the operation small portions may be overlooked and left behind as a result of tears of the cyst walls, which are very thin in many cases. The consequence of such a mishap is the formation of a fistula and a delay of healing until the bits which have been left behind have been removed. In all of the three cases reported the function of the eyeball was preserved, and the visual fields, which at first were considerably contracted, regained normal limits as the vision improved.

WRIGHT (77, **Tumor of orbit of doubtful origin**) described a tumor mass located in the superior temporal quadrant of the orbital roof. The ground was composed of endothelial cells with a very open intervening reticulum, without continuity in the burrowing tissue. These cells are in many places continuous with the walls of the smaller vessels which are composed of several layers of endothelium. Scattered throughout were lymphocytes, red blood cells and eosinophiles. No microörganisms were found. There was some signs of mitosis and giant cells in this conglomeration. ALLING.

In the case reported by ELSCHNIG (65, **Pseudotumor in the orbit caused by an encapsulated splinter of wood**) the patient, 21 years old, had been wounded just below the right eye with a stick of wood twelve years before. The wound healed smoothly without any special trouble. During the last few months he had noticed a slowly growing tumor of the lower lid which had displaced the eyeball upward. The tumor was removed and found to consist of granulation tissue and

foreign-body giant cells about an encapsulated splinter of wood. The case seems to be noteworthy because the splinter did not cause an abscess in the orbit.

ENGELKING (66, **Symmetrical tuberculomata of the orbit**) reports the case of a woman 51 years old who, for four months, had noticed slowly growing, painless tumors on the two lower lids. The skin over the growths was normally movable. The tumors extended into the orbits. The transition folds of conjunctiva were somewhat thickened, the lacrimal glands not involved. Internal organs normal; no syphilis or tuberculosis; lymphocytosis with a little eosinophilia. Clinical diagnosis—Lymphomatosis. Tumors shelled out readily; smooth healing; no recurrence at the end of three months. The results of the pathological study of the tumors are given in rather ambiguous words: Tuberculosis of the lymphatic tissue no bacilli demonstrable. Ocular and peritoneal inoculations of rabbits negative.

To remove malignant tumors from the orbit FILATOW (67, **Removal of malignant tumors from the orbit**) recommends a modification of an operation said by him to have been published by Golowin. He incises the skin about the orbit in the form of a square and undermines the adjacent tissue. After dissecting up all the soft parts of the orbit subperiosteally, the various bones about the orbit are chiselled off and the accessory sinuses opened, so that the latter may be cleaned out if necessary. The mass composed of the bones and soft parts is then seized with large forceps, drawn out of the cavity and cut away at the apex of the orbit. The wound cavity is now lined with such sound parts of the lids as may have been preserved, or with sound tissue from the neighborhood of the orbit. The nose and pharynx are tamponed. The tampon in the pharynx is removed six to eight hours after the operation. The dressing to be changed every third day, the tampon in the nose being drawn a little further out each time. After the sixth day the tampon through the nose is renewed. The writer says that he has operated on four cases in this manner with good results.

FRANKLIN and CORDES (68, **A case of orbital lymphangioma**) report a case in which the cyst was removed by Kronlein resection without enucleation of the eye. They believe

lymphangioma is a misnomer for this kind of a mass and suggest that the term lymphangiectasis be used instead.

ALLING

DI MARZIO (73, **Cavernous angioma of the orbit**) describes four cases of this nature which were operated on with preservation of the eyes by Cirincione's orbitotomy. The tumors varied in size, each composed of a fibrous capsule containing lacunæ filled with blood separated by connective tissue septa. In each case the mobility of the eye remained intact after he operation, the vision was saved, and indeed improved in two of the cases. In one eye with choked disk and central scotoma, the central disturbance disappeared and the papilla regained its normal appearance. In the fourth case, suffering from secondary optic atrophy, the eyeball was preserved with its normal mobility.

RIDLEY (75, **Intraorbital neoplasm**) observed a typical giant cell sarcoma in a woman 61 years old. The tumor came from the inner side of the orbit and had walled in the optic nerve and the posterior segment of the eyeball. After the orbit had been exenterated a little prominence was noticed in the sphenoidal fissure, but this disappeared completely under the influence of radium. No pathological condition of the accessory sinuses could be detected by the X-ray and there was no swelling of the glands. The clinical symptoms were the well known increasing exophthalmos with loss of vision.

STIEREN (76A, **Melanosarcoma of choroid with extension to orbit**) reports a case which was treated by radium after a generous enucleation including all the involved orbital tissue was done. There has been no local recurrence or distant metastasis after almost three years. ALLING.

DELORD (64, **A case of sarcomatous metastasis in the meninges with bilateral exophthalmos and sinus thrombosis**) observed the following symptoms in a 3-year-old child. Bilateral exophthalmos, total inhibition of ocular movements, disturbances of sensation, circulatory stasis of the veins in consequence of the obstruction to circulation in the cavernous sinus, and disturbances of motility from compression of the nerves and vessels passing over the sphenoidal fissure. Some days later an enormous œdema appeared in the temporal region, spread to the pharynx, and finally to the entire face.

These symptoms together gave the characteristic picture of a complete occlusion of the circulation in the sinus. At the autopsy tumors were found in the mesentery, in both kidneys, and a fungus of the dura mater with multiple sinus thromboses. These tumors had the same structure and were pronounced to be atypical lymphocytomata.

MAGGIORE (72, **Thrombophlebitis ophthalmo-cavernosa**) describes a case in which an abscess of the upper lip developed from the sting of a wasp, led to a septic thrombosis of the orbital veins with involvement of the cavernous sinus, and death. The conditions present in both eyes were: Œdema of the lids, inflammatory exophthalmos, thickening and œdema of the cornea, infiltration and thickening of the iris, choked disk, and detachment of the retina with an exudation rich in fibrin in the meshes of the stroma of the choroid. The retrobulbar fat was infiltrated with pus. The walls of the veins, the nerve sheaths, and the interstitial tissue of the optic nerve were densely infiltrated with little cells. The rapidity of the spread of the infection was remarkable.

YERGER (78, **Exophthalmos a complication of nasal sinusitis**) from a study of seven cases finds that the path of infection from the nasal sinuses is most usually from a necrosis in the common bony wall of the sinus and orbit. From a review of the literature he notes that exophthalmos due to suppurative sinusitis is invariably unilateral and that the most common orbital complication is orbital cellulitis and abscess. It is also noteworthy that while suppurative nasal sinusitis is relatively common, yet the incidence of orbital phlegmon is infrequent and exophthalmos is rare. ALLING.

RING (76, **Orbital cavernous hæmangioma with pulsating exophthalmos. Removal by electrocoagulation.**) described the technic of the electrocoagulation operation by which the mass was shelled out. The orbital growth was recognized early in life and treated at first by chemical cauterization and later by ligation of common carotid artery. After a period of quiescence the eye tissue broke down and the proptosis became more marked. At this time she was seen by Ring. ALLING.

LOCKE (70, **Intracranial arteriovenous aneurysm or pulsating exophthalmos**) describes three cases of his own in detail

and studies the ætiology, pathology, and treatment of pulsating exophthalmos in 588 reported cases. In 76, 84% of the cases reported trauma was the ætiological factor. In summarizing the pathology practically all traumatic types are due to an intracranial arteriovenous communication. A little over one half of the cases of the spontaneous type could be attributed to a vascular intercommunication, about one quarter being caused by tumor and the remainder by simple aneurysm. He goes into detail about the treatment of this condition.

ALLING.

KUBIK (69, **The influence of exophthalmos upon the action of the obliques**) made his observations on a case of exophthalmic goitre in whom the exophthalmos was very marked. The depressing component of the superior oblique becomes nil when the point of rotation of the eyeball and the insertion of the muscle and the trochlea lie in the same plane. As the exophthalmos increases the superior oblique comes to act as an elevator. The abducent action decreases gradually until it becomes nil when the exophthalmos is maximal. The rolling effect is increased with the exophthalmos and reaches its maximum when the line joining the trochlea and the place of insertion of the muscle is perpendicular to the plane of the axis of the eye. The opposite holds in all respects for the inferior oblique.

LODATO (71, **Radiologic investigation of compression atrophy of the optic nerve**) describes two cases of descending optic atrophy, the cause of which was determined by the X-ray to be in one case a tuberculous periostitis in the anterior part of the sella turcica, in the other a gummatous disease of the ethmoid and sphenoid bones.

MORELLI (74, **A case of menstrual exophthalmos**) gives the history of a girl 13 years old who had adenoids, chronic tonsillitis, and a tendency to nosebleed. Some time after the adenoids had been removed exophthalmos of the right eye appeared, accompanied by slight pain. These disappeared after a few days, but recurred regularly every four weeks. The eye condition was normal, the tension was not elevated, the fundus and vision were not changed, and no inflammation could be found in the accessory sinuses. In the fifth and sixth attacks the exophthalmos was accompanied by head-

ache, nausea and a feeling of heat; this lasted five or ten days when nosebleed and hæmoptysis set in and the troubles disappeared. Ovarian preparations were prescribed and at the end of four weeks the first menstruation appeared instead of the expected troubles. From this time on menstruation was regular, and there was no reappearance of the exophthalmos and associated symptoms.

V. THE LIDS AND LACRIMAL ORGANS.

79. BEACH, S. JUDD. **Œdema of the eyelids.** *Journal A. M. A.*, July 5, 1924.

80. BLATT. **Primary anthrax of the lids.** *Archiv f. Ophthalm.*, cxi., p. 60.

81. CONTINO. **Histogenesis of contagious molluscum of the lids.** *Annali di Ottalmologia e clinica oculistica*, Nos. 6–8, 1923.

82. GABRIELIDES. **Bloody tears.** *Annales d'oculistique*, clx., p. 705.

83. ISHIKAWA. **Pseudoleukæmic changes in the skin and the eyelid.** *Zentralbl. f. Ophthalm u Grenzgebiete*, ix., p. 283.

84. KRAMER. **Recurrent purulent dacryops.** *Zeitschr. f. Augenheilkunde*, li., p. 337.

85. KRAUPA. **Gifford's sign.** *Klin. Monatsbl. f. Augenheilkunde*, xxi., p. 196.

86. KUMMER. **Radium treatment of some diseases of the lids.** *Zeitschr. f. Augenheilkunde*, xlix., p. 353.

87. LEXER. **Operation for ptosis.** *Klin. Monatsbl. f. Augenheilkunde*, lxx., p. 464.

88. LOEB. **Neurofibroma of the upper lid.** *Archiv. f. Augenheilkunde*, xciii., p. 74.

89. MENDE. **Treatment of phlegmonous dacryocystitis.** *Klin. Monatsbl. f. Augenheilkunde*, lxxi., p. 212.

90. MORETTI. **New operation for entropion of the lower lid.** *Bolletino d'oculistica*, May, 1922.

91. NOBL. **Treatment of xanthomatosis of the lid.** *Med. Klin.*, xix., p. 1631.

92. RIZZO. **Perithelial sarcoma of the tarsus.** *Annali di Ottalmologia e Clinica oculistica*, April-May, 1923.

93. RIZZO. **A myxomatous polyp of the lacrimal sac.** *Bolletino d'occulistica*, April, 1923.

94. RUSCHE. **Gebb's treatment of purulent of dacryocystitis.** *Klin. Monatsblätter f. Augenheilkunde*, lxxi., p. 226.

95. STELLA-GANGI. **Trachoma of the lacrimal gland.** *Bolletino d'ocul.* August, 1923.

96. TRISTAINO. **Two cases of acute dacryoadenitis.** *Ibid.*, March, 1923.

BEACH (79, **Œdema of the eyelids**) mentions the various hypotheses which have been proposed to explain the cause of the anomalous œdema. He believes that the ætiology is, of course, only speculative, but pathologically we are dealing with a true œdema of the subcutaneous tissue. In treatment, efforts should be directed toward strengthening immunity.

ALLING.

BLATT (80, **Primary anthrax of the lids**) reports ten cases. The primary lesion on the lid does not present the typical appearance of the anthrax carbuncle, because the loose skin and subcutaneous tissue of the lid affords little resistance to the extension of the lesion. Four stages may be distinguished in its development. First, itching and redness with swelling of the lid and of the lymphatic glands; second, necrosis of the superficial and deep layers of the skin of the lid; third, the casting off of the necrotic part at a purulent line of demarcation; fourth, cleaning up of the ulcer or the involvement of the eyeball in the gangrene. The infection had been carried by flies. The time of incubation of the disease was four to five days. To improve the general condition Blatt gave neosalvarsan intravenously. This did not influence the disease in the eye, which received only conservative treatment.

CONTINO (81, **Histogenesis of contagious molluscum of the lids**) says that molluscum contagiosum consists of a hyperplastic production of epithelium induced by a specific agent, which on the one hand exerts a proliferative influence upon the germinal stratum, and on the other induces degenerative changes in the newly formed cells, which, together with the physiological process of cornification, produces the characteristic formation. These growths start only in the skin, and are found with a certain regularity at the mouths of the hair follicles, the walls of which offer less resistance to the entrance of the virus. The beginning of the growth is characterized by a circumscribed cell proliferation in the stratum germinale, which sinks into the dermal tissue as a solid, conelike sprout, surrounded by a dense infiltrate of small cells. The solid cone gradually becomes canalized through central breaking down and clothed with horny cells through cornification. In the lower part of the sprout, the germ center, vesicular cells are to be seen in this stage of development, the

endoplasm of which is at first fluid, later clotted and finally cornified; they form in their entirety the central molluscum corpuscle. The molluscum increases its size by a broadening of the sprout and the sending out of numerous offshoots, which likewise become canalized, until it finally is transformed by the stretching of the lumina of the canals and laceration of the intermediate walls into a large cyst with cornified lateral walls which contain lamellæ of horn and molluscum corpuscles.

ISHIKAWA (83, **Pseudoleukæmic changes in the skin of the eyelid**) reports the case of a patient 31 years old who had typical acute aleukæmic lymphadenosis. All four eyelids were swollen and firmly elastic. Uneven tumors and infiltrates were to be seen on the temples, cheeks, chin and scalp. A tumor as large as a walnut was on the sternum and on the right side of the abdomen. Similar tumors were transiently present in other parts of the body. The patient died of influenza. The blood picture showed 960,000 erythrocytes, 720,000 leucocytes, lymphocytes 70–80 per cent. myeloid cells 20–30 per cent. for the most part polynuclear, neutrophile leucocytes. The changes in the skin were caused by macrolymphocytes. The areas of the skin which appeared to be normal also showed lymphatic cell deposits about the vessels, glands, nerves, and in the subcutaneous fat. These little foci are, in the opinion of the writer the starting points of the skin tumors. The corium remains free. The aleukæmia depends on the obstacle to the escape of the leucocytes from the glands, spleen, and bony marrow. The tumor of the lid was caused chiefly by infiltration of the lacrimal gland, but almost all the other parts of the lid were involved. The swelling of the lower lid was produced by a heteroplastic lymphoid transformation of the connective tissue. Orbital tumors are further advanced pseudoleukæmic changes of the lid. The upper transition fold was protruded by infiltration of the lymph follicles, Krause's glands, and the lacrimal gland. The eye exhibited detachment of the retina, little hemorrhages in the retina, and a deposit of lymphocytes in the choroid.

Gifford described in 1906 as an initial symptom of exophthalmic goiter a difficult eversion of the upper lid. KRAUPA (85, **Gifford's sign**) doubts the infallibility of this symptom as a sign of a forthcoming onset of exophthalmic goiter, and

reports a case in which eversion of the upper lid was very difficult because of the condition of its skin, although no other sign of exophthalmic goiter was present, or has since appeared.

KUMMER (86, **Radium treatment of some diseases of the lids**) says that angiomata should receive this treatment only in an early stage, and that it is better not to use it when they have become ulcerated as in such cases the ulceration is apt to be made worse. The more prominent the angioma, the better the prognosis from the irradiation. Teleangiectasies and spider nævi respond better to the thermocautery electrolysis, and scarification. The effect of radium in nævus flammeus is questionable. Good results are obtained in ninety-five per cent. of the cases of cavernous cutaneous angiomata treated with small doses of gamma rays. Sub-cutaneous cavernous angiomata and lymphangiomata require a long treatment with small doses, which cure seventy-five per cent. Lupus vulgaris is less suited for radium; the hypertrophic form reacts the best. Fungoid tuberculous lacrimal sacs respond very well. Excellent results are obtained in epithelioma of the skin, which should always receive radium. Xanthelasma and pigmented nævi are not suitable for this treatment. If the proper technique is observed, an almost invisible scar is left, after any of these lesions has been cured by radium. Deep-seated painful carcinomata may be relieved by radium, but a cure is not to be expected, and a long treatment is necessary. The eye should be protected from the rays by a lead plate.

LEXER (87, **Operation for ptosis**) lifts the lid by means of two narrow strips of fascia which converge downward. Two incisions scarcely 1*cm* long are made in the eyebrow, one in the outer, the other in the inner third down to the muscle. From these two canals are formed beneath the skin of the upper lid nearly to the margin of the latter. Some 3*mm* above the margin of the lid, at the lower ends of these canals, two little incisions are made through the skin. A strip of fascia, not over half a centimeter wide, is then passed through each canal, the lower ends secured at the little wounds in the lid. As the strips of fascia contract the lid must be raised only partially, to what he calls the "tired position,"

and then the upper ends of the strips are attached to the occipito-frontalis.

LOEB (88, **Neurofibroma of the upper lid**) observed a case of this nature in a girl 15 years old. The tumor had first appeared three years before. Its removal was difficult, as it was necessary to preserve the levator. Examination of sections with the microscope revealed many nerve fibres with exuberant perineurium between bundles of connective tissue, which appeared to be secondarily broken down nerve bundles.

MORETTI (90, **New operation for entropion of the lower lid**) makes an intermarginal incision and then fastens into it a flap of skin from the lower lid. The flap is as long as the incision, is 2½*mm* broad, and has its pedicle at the outer canthus.

NOBL (91, **Treatment of xanthomatosis of the lid**) recommends painting with trichloracetic acid as superior to any other method of treatment, mentioning among others excision X-rays, radium, electrolysis, carbonic acid snow, and ignipuncture. He says that the painting is not painful and acts through the penetration of the acid through the somewhat more easily vulnerable epithelium over the xanthelasma. The delicate scab falls off in from eight to fourteen days and leaves neither cosmetic or functional trouble. After the healing is complete one usually sees no fault; frequently the place of the former disease can be recognized through a slight depression of the skin. Recurrences are rare and can easily be controlled by a renewal of the painting.

RIZZO (92, **Perithelial sarcoma of the tarsus**) observed a tumor of this nature, as large as a hazel nut, attached by a broad base to the tarsus of the right upper lid of a woman 66 years old. The tumor was removed with the tarsus and the neighboring skin of the lid, the defect was closed by a plastic operation, and a perfect recovery followed. No recurrence had appeared at the time of writing.

GABRIELIDES (82, **Bloody tears**) reports his observations in two cases of this nature. One patient was a widow 36 years old who, independently of time or place, since the early days of her widowhood had bloody tears from time to time, sometimes from one eye, sometimes from the other, sometimes from both. From her statements there seemed to be no connection with menstruation. The patient was of a very sensitive

nature and wept frequently and copiously, but as soon as the bloody tears appeared she lost the desire to weep, experienced an internal oppression, and was seized with a sudden desire to leave the place where she was at the moment. Frequently she had buccal hemorrhages. No pathological condition could be found in the eyes or in the general bodily condition. The other patient was a girl of 14, in whom bloody tears were observed several times a day almost daily during the absence of the menses. Simulation was excluded. Gabrielides had an opportunity to observe an attack in his office. The conjunctivæ were reddened, the vessels tensely distended. On the upper tarsal conjunctiva were about twenty small distinctly visible flecks of blood; these were absent along the free margin of the lid and at the ends of the tarsus. The lacrimal glands were normal. Bloody tears were mentioned first by Euripides. Medical attention was first called to them in the beginning of the seventeenth century, but the number of cases thus far observed is very small. Connection with the endocrine functions is suggested.

KRAMER (84, **Recurrent purulent dacryops**) reports the case of a patient who had a noninflammatory swelling of the right upper lid at the outer canthus. Pus escaped from the tumor when pressure was made upon the upper lid. The lacrimal gland was removed, and its lower part found to be transformed into a cyst. Kramer thinks that the cause probably was an imperfectly emptied purulent dacryoadenitis.

TRISTAINO (96, **Two cases of acute dacryoadenitis**) observed in a man 27 years after an attack ot rheumatism, an inflammatory œdema of both upper lids with fever and pain. A very tender tumor could be felt in the region of the lacrimal gland, the lacrimal secretion was stopped, the eyeballs were dislocated inward and downward, and their abduction was limited. Diplopia could be demonstrated by means of colored glasses, the vision was reduced to 0.5, although there was no error of refraction. There was no exophthalmos. The same disease appeared on one side in a 6-year-old child after an attack of measles. In this case the inflammation extended into the retrobulbar tissue and caused exophthalmos. Both patients recovered under local treatment within a couple of weeks, with no permanent injury.

STELLA-GANGI (95, **Trachoma of the lacrimal gland**) observed a trachoma of the lacrimal gland in a man 52 years old with cicatricial trachoma. The disease began with a painful swelling of the lacrimal gland, which finally had to be removed as a hard elastic tumor as large as a hazel nut. Investigation showed the glandular tissue was replaced by a degenerative tissue divided by fibrous septa. This degenerative tissue consisted of foci of infiltration with lymphocytes with numerous central trachoma follicles.

v. MENDE (89, **Treatment of phlegmonous dacryocystitis**) claims that a phlegmonous dacryocystitis may be influenced so favorably by a pressure bandage that an incision does not appear to be necessary. After the inflammation has subsided he usually excises the lacrimal sac.

RIZZO (93, **A myxomatous polyp of the lacrimal sac**) found this growth in a lacrimal sac which he had excised from a 48-year-old patient on account of chronic dacryocystitis. It was a bean-shaped tumor measuring 5 by 3*mm*, a gray white, firm body with a smooth surface and a homogeneous, gray white, fluid content. The tissue consisted of polymorphous cells with a homogeneous intermediate substance, abundant vascularization, and infiltrates of small cells.

RUSCHE (94, **Gebb's treatment of purulent dacryocystitis**) tried the method suggested by Gebb of injecting certain ointments through the uncut canaliculi with a blunt canula. The effect was comparatively good upon the purulent secretion, which disappeared after two or three injections, but the bacteriological condition and the epiphora remained uninfluenced. The injections were for the most part free from pain. From three to twelve commonly suffice to cure. Twice they led to phlegmons. This procedure seems suited for cases in which patients refuse to be operated on.

(*To be Continued.*)

CORRESPONDENCE.

Technic of Intracapsular Extraction.

To the Editor:

I have read with interest the article by Col. Henry Smith in a recent issue of the Archives, describing a new technic for the expression of so-called hard cataracts in their capsules. If I may, I should like to invite your attention to the fact that the method of tumbling hard cataracts as well as soft ones is the one taught me in 1922 by Dr. Mathra Das, a native surgeon in charge of the hospital at Moga, District of Ferozepore, Punjab, India. While I was at his Clinic in the Punjab I observed him remove about 3,000 cataracts in their capsules. He took great care to explain that his technic differed from the classical Smith in that he tumbled them all instead of only the soft ones. His procedure in hard cataracts is as follows:—

After cocainizing the eye, the lashes are clipped and conjunctival sac irrigated with weak bichloride solution. A spring speculum is inserted. The operator makes corneal section while the assistant holds the brow with one hand and steadies the speculum with the other. The section is a full 180 degrees with puncture and counter puncture about *1mm* lateral to limbus. It is finished above about *2mm* in the clear cornea. After a small complete iridectomy is made above, the speculum is removed. The operator then pulls the brow back with his left thumb and delivers the lens with a Smith hook in the right hand while the assistant pulls the upper lid away from the eye with the large hook in one hand and gently depresses the lower lid with the other. The lens is delivered by first dislocating it by pressure with the Smith hook about *2mm* below the limbus at six o'clock causing its lower edge

to tilt forward. The hook is carried steadily upward under the lens until it is lifted out or rolled off the eye, the cornea having been folded under. After repositing the iris by stroking the angles of the wound the eye is closed and bandaged for seven days.

I feel that Dr. Das should be given credit for developing this method of delivering hard cataracts which is certainly superior to the old technic and which he had employed in thousands of cases before Col. Smith's return to India.

LELAND L. BULL, M.D.

SEATTLE, WASHINGTON,
November 26, 1926.

NOTICES.

October, 1926.

At a general meeting of the Convention of English-speaking Ophthalmological Societies held in London in July, 1925, it was resolved "that this Convention of English-Speaking Ophthalmological Societies hereby empowers its President, Mr. E. Treacher Collins, to appoint a standing committee of five, of which he shall be chairman. The duty of this committee shall be to obtain such co-operation as is possible from representatives appointed by various ophthalmological societies for the promotion of international ophthalmological congresses." It was also resolved that other matters of international ophthalmological interest should be referred to the international committee, when constituted.

The committee of five, constituted in accordance with the above resolution, considers that the time has now come to take further steps to form the larger representative international committee suggested. It is desired that each nation should be represented by two delegates nominated by arrangement between the principal ophthalmological societies of the nation, or, in the case where no national ophthalmological society exists, by representative ophthalmologists. The meetings of the committee will be held at The Hague, in July, 1927.

The committee will be glad to receive the names and addresses of the two delegates selected to represent your country as soon as you are able to make arrangements for their nomination.

E. Treacher Collins,
Edward Jackson,
John Herbert Parsons,
G. E. de Schweinitz.
Leslie Paton.

PRELIMINARY NOTICE.

THE OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM.

17th November, 1926.

The Annual Congress of the Ophthalmological Society of the United Kingdom will be held in Liverpool on Thursday, Friday and Saturday, April 28th, 29th and 30th, 1927. The Subjects for Discussion are:

1. "The Etiology and Treatment of Hypopyon Ulcer."
2. "Nystagmus, *Excluding* Miners' Nystagmus."

Members who wish to take part in the discussions or propose to read papers should communicate with

MR. H. P. GIBB,
53, Harley Street, London, W. 1.
CHARLES GOULDEN, H. P. GIBB } *Secretaries.*